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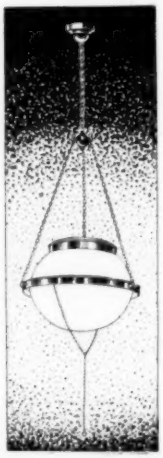

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By Daniel T. Davies, M.D., B.Sc. Wales,
M.R.C.P. Lond.,
ASSISTANT PHYSICIAN TO THE ROYAL FREE HOSPITAL.
(*From the Middlesex and Royal Free Hospitals.*)

In this article the name simple achlorhydric anæmia, first suggested by Witts,[1] will be used to mean that form of chronic and simple anæmia found in middle-aged women accompanied by achlorhydria. Many other titles have been offered, such as cryptogenetic achylic chloranæmia,[2] pseudo-pernicious anæmia,[3] hypochromatic anæmia,[4] and idiopathic secondary anæmia,[5] but the first title seems to be the most suitable, for it draws attention to the two salient features—a simple anæmia and an achlorhydria—without in any way confusing the condition with pernicious anæmia. Attention was first drawn to it by Faber [6] in 1914, and several papers by continental observers have since emphasised the distinctiveness of the syndrome and its treatment with iron. The credit of rousing English interest in this disorder is due, however, to Witts, who has recorded his studies in recent publications.[1 7] American work on the subject is seen in the writings of Mills,[5] Watkins,[8] Waugh,[4] and Mettier and Minot.[9]

Although the condition appears to be comparatively common, it has not been widely recognised as a distinct entity. There are many possible reasons for this lack of recognition, among which the chronicity and mildness of the anæmia, the absence of any acute form, and the prominent digestive symptoms may be cited. Indeed the multiplicity of the symptoms complained of, coupled with the listlessness shown by a chronically anæmic woman, is hardly likely to incite clinical enthusiasm. Nevertheless, once the syndrome is recognised as an entity, it is found to be a frequent source of ill-health in women, and one in which therapeutics can materially aid the patient to a return of well-being. In addition, a study of this anæmia throws some further light on the functions of the healthy stomach and its secretion.

The Clinical Picture.

The disease is far more commonly found in women than in men. In Witts' series of 50 cases, 49 were females. In my collection of 55 only four were males. Mills and Waugh also remark on the predominance of the female. In my series the maximum age incidence is between 35 and 50 years—the youngest 30, and the eldest 65. It is therefore a disease of adult womanhood. The duration of the anæmia is usually of many years, five and ten years being common. The additional gastro-intestinal symptoms may also be of equally long duration or even longer. The patient, having suffered from flatulence and pallor for so many years, often fails to fix any date to the first appearance of the symptoms—"having always been pale" and "having always suffered from indigestion."

The symptoms can be divided into two natural groups—those directly due to the anæmia and those due to the digestive disturbances. The symptoms due to the former are in no way different from those of other anæmic states, with the one exception that, as the hæmoglobin is diminished to a much greater extent than the red cells, dyspnœa may become such a prominent symptom as to incapacitate a person from daily work and yet the red cells number above four million. One of the males of the series was a blacksmith, quite incapable of performing his work, although his red cells stood at five million, but his hæmoglobin was only 50 per cent. This state contrasts with that seen in a patient with pernicious anæmia, who is usually at work until the red cells have fallen below three millions.

The alimentary symptoms, although of varying degree, are present almost without exception. Discomfort and fullness after food, a poor appetite, and flatulence are common. These, again, are of long-standing and may dominate the picture for a number of years. The anorexia may be more marked in the morning and improve towards evening. Discomfort is worse after meat and least after carbohydrate foods. In consequence the diet becomes one of bread, potatoes, milk puddings, and tea, the patient being led by her symptoms to believe that other foods are definitely injurious. Many of the patients, when asked, remarked on their loss of desire for any change in diet; they had not touched meat for years, and they were satisfied if they ingested small and frequent carbohydrate feeds.

Symptoms suggestive of visceroptosis can often be elicited from these patients and, indeed, some have resorted to abdominal supports. Diarrhœa was not complained of by any of the observed cases. Although the digestive symptoms are of varying severity they appear to be far more constant than in pernicious anæmia and far more taxing to the patient. They have commenced in such an insidious manner that it is difficult for the patient to give any exact information about their onset.

The appearance of the patient is characteristic in the fully developed case, for the complexion has a peculiar sallowness—almost a lemon tint, as seen in pernicious anæmia—and the sclerotics show a bluish colour; the picture thus does not resemble secondary anæmia from hæmorrhage. The patients are often underweight, and the wrinkles, and folds of inelastic skin contrast with the rotound appearance in pernicious anæmia. Many of my patients were grossly underweight—some only 6 st.—another result of their poor intake of food. The patients often exhibit fissures and painful cracks at the angles of the mouth, and this is accompanied by a well-marked atrophic condition of the tongue mucosa. The more severe the anæmia, the more angry and red does the bald tongue appear, but it is uncommon to obtain complaints of soreness of the tongue. In a few the tongue shows less marked changes, the only observable lesion being atrophy of the papillæ. In this series only five out of 55 showed no tongue changes. The high frequency of a glazed tongue is therefore a valuable diagnostic feature, and more constant in this condition than in pernicious anæmia.

Another characteristic abnormality exhibited by these patients is seen in the nails, which may be unduly brittle, tend to split longitudinally, are commonly flat and sometimes "spoon-shaped"—"koilonychia." Kaznelson [10] drew attention to these nail changes, and emphasised the improvement obtained on adequate treatment. Four of the present series showed the extreme spoon-shaped changes, while 31 complained of excessive brittleness and tenderness, and 26 showed flattening. These nail changes seem to be peculiar to this disease and are not found in pernicious anæmia.

When the anæmia is marked the spleen is often palpable, but not greatly enlarged. With improvement

in the anæmia the spleen returns to its normal size. The abdominal wall is atonic and inelastic, and frequently the abdominal aorta can be palpated with undue ease.

The anæmia is more obvious during and after a pregnancy, to improve somewhat later. One of my patients required active treatment during her pregnancies in 1925, 1927, and 1929, but in between the pregnancies, although pale, she was able to do her work. Another gave a similar history during two pregnancies; she improved on their termination, but remained pale. Menstrual irregularities are also recorded; six women complained of amenorrhœa and four of excessive bleeding. So severe was the bleeding in two that radium treatment was contemplated. With the improvement in the anæmia, these patients also showed an improvement in the menstrual cycle. Two of the women gave histories of hæmatemesis, one 23 years previously, and the other 11 years previously, but the majority gave no history of abnormal hæmorrhage, so that the anæmia cannot be accounted for simply by loss of blood.

THE BLOOD.

While there is generally a marked reduction in the hæmoglobin content the red cells are often surprisingly normal. When the hæmoglobin is 40 per cent. or less, it is quite common to find the red cells at four million. The colour-index is therefore much below unity. The average hæmoglobin content of the present series was 52 per cent. and the average red cell count 3·9 million. The lowest hæmoglobin percentage was 25 and the lowest red cell count 2·58 million. No marked abnormality is found in the red cells other than those common to secondary anæmia. Some show a low average diameter, such as 6·9 μ, while others are normal. Witts [1] frequently found microcytosis, but a few patients may show the reverse condition—macrocytosis and a high average red cell diameter. While the majority of my patients showed no abnormal features in the blood films, except those of secondary anæmia, three—two male and one female—showed an average red cell diameter of 7·8 μ and macrocytes. The three were over 55 years of age and are probably examples of the intermediary type between this syndrome and pernicious anæmia.

Some cases show a very minor degree of anæmia; four of this series of 55 showed hæmoglobin values above 70 per cent., although the other features of the disorder were quite manifest on examination. This is of some importance, for it shows that the tongue and nail changes, accompanied as they are by the symptoms of indigestion, may precede the development of anæmia; but the anæmia in such a case may develop rapidly as a result of any aggravating factor—e.g., menorrhagia or pregnancy.

The white cell count in this series varied between 3000 and 7600, counts of 3000–5000 being usual in the more anæmic cases. With improvement in the blood hæmoglobin, the white cells, if previously low, increase to a more normal figure. None of the patients had a leucocytosis.

There is no evidence of increased hæmolysis, for the serum bilirubin is always low and in consequence the serum is pale, a finding which suggested the name "hypochromatic anæmia" in contrast to hyperchromatic or pernicious anæmia.

The blood examination shows, therefore, evidence of a secondary and simple anæmia, the result of hæmoglobin deficiency, which is not due either to blood loss or to excessive hæmolysis, and which is not accompanied by any evidence of a primary marrow disease.

THE GASTRIC SECRETION.

The gastric secretion is of more than usual interest in this condition; indeed the achlorhydria that is commonly found may be, as in pernicious anæmia, the basal and underlying factor in the development of the syndrome. On ordinary fractional analysis, in addition to the absence of free hydrochloric acid, two other features are observed: rapid emptying-rate and increased viscosity of the juice. Emptying-rates of half an hour to one and a half hours are usual, as in all achlorhydric states. Owing to the increased mucus content it is difficult to aspirate the juice. These three findings, the absence of free hydrochloric acid, the increased mucus content, and the rapid emptying-rate, are indicative of a pathological state of the stomach, and are in keeping with the marked symptoms of indigestion.

A clearer understanding of the extent of this deranged secretion is obtained when the juices are examined, before and after the subcutaneous injection of histamine. Of 24 patients of the series who were submitted to the injection of histamine, 12 showed free hydrochloric acid in the samples of juice withdrawn after injection. A further four showed a fall in pH, but not of such a concentration as to be titratable as free hydrochloric acid. In other words, 16 of these 24 patients were not completely incapable of secreting hydrochloric acid, but did so only under stimulation; while eight failed to secrete acid even under stimulation.

Pepsin was demonstrated in 19 out of 25 patients, either in the resting juice or in the samples of juice after histamine injection. In no case was the pepsin secretion found to be as much in health (250–500 units), the average figure being only 70 units. Six showed no pepsin secretion, as well as no acid secretion—true achylia.

A typical result of histamine stimulation is shown in the following figures (Table I.) from a woman aged 44, with a previous achlorhydria on ordinary gastric analysis and exhibiting a mild degree of anæmia (red cells 3·76 million; hæmoglobin 46 per cent.)

TABLE I.

—	Resting juice.	After histamine injection.	
		10 min.	20 min.
Volume	20 c.cm.	15 c.cm.	12 c.cm.
Appearance	Cloudy.	Clear.	Clear.
Mucus	+ + +	+ +	+ +
pH	7	7	7·3
Hydrochloric acid	0	0	15
Pepsin	4	64	120

Such a response is obtained in the larger number examined, and presumably is a fair index of the state of the gastric secretion of the majority of the patients. This secretion, with its diminished hydrochloric acid and pepsin content and its excessive mucus, would be consonant with "gastritis."

It is interesting to compare this state of affairs with that seen in pernicious anæmia. In both, an ordinary gastric analysis would disclose an achlorhydria, but, while the achlorhydria in this condition is often only "apparent" or "relative," it is "absolute" in pernicious anæmia even when the patient has returned to health—i.e., the reaction of the stomach juice remains constant. Again, in this syndrome pepsin is still being secreted while there is an absence of pepsin in pernicious anæmia—a true achylia. Finally, mucus, a feature so prominent in the juices of this simple anæmia, is absent in pernicious anæmia.

Examination of the gastric secretion in the syndrome of simple achlorhydric anæmia therefore discloses a marked degree of gastric disturbance, but not of such a severe grade as in pernicious anæmia. The fact that the symptoms are more obvious and distressing is probably accounted for by the fact that the juices are so viscid.

Contributory Factors in Development of the Disorder.

Although the cause of the disease has not been proved, a consideration of the clinical features is highly suggestive of a nutritional disorder. The disease is always chronic, no acute form having been described; it is amenable to iron therapy, and therefore cannot be a primary blood disease; it is not materially influenced by the administration of stomach or liver extract, and therefore the deficiency present in this syndrome cannot be of the same nature as in pernicious anæmia. The two important positive features which may contribute to the genesis of this disorder are the diet and the gastric secretion.

The most outstanding feature is the lack of meat ingestion. Many of the patients, on questioning, say that they have not taken any red meat for years, for the obvious reason that they suffer discomfort from meat eating. The same applies to green vegetables. The diet is mainly composed of concentrated starchy foods, of which bread is the chief. Fish is taken without any discomfort. It must be remembered that this diet, poor both in protein and in mineral elements, has been the mainstay of these patients for years. Such a diet is analogous to the diet of tropical countries where anæmia is common.

A similar anæmia produced by feeding polished rice to rats was found by Hart [11] to be as amenable to beef as to liver for re-establishing the hæmoglobin. Bunge,[12] who studied the iron content of the common articles of food, drew attention many years ago to the poor iron content of cereals, and deplored the fact that in the production of fine wheat flour the iron contained in the wheat had been diminished to a fifth of its original value. His emphatic words on the mistakes of treatment adopted for anæmic women in those days are worth quoting: "We may take as an instance the poor bloodless seamstress who eats white bread and drinks tea. With the aid of all our chemical knowledge, we could not give to animals which we wished to make anæmic any food with less iron in it." Milk, rice, and potatoes are all poor in iron, and while red meat contains 17–20 mg. of iron per 100 g. of dried substance, wheat flour contains only 1·6 mg.

Knowing, therefore, that the patients exhibiting this syndrome partake of such a diet for so many years it is justifiable to conclude that their iron intake is poor and liable to result in an iron deficiency. But although a dietetic abnormality is exhibited by the majority, it must be conceded that such an unbalanced diet does not invariably result in an anæmia. On such a restricted diet many patients with a gastric ulcer retain normal blood figures. However, in this syndrome, in addition to the abnormal diet, there is a highly abnormal gastric secretion. It does not appear to be of essential importance to discuss which is the primary condition; it may well be that the gastritis is the initial lesion and the abnormal diet the sequel, but, be this as it may, it seems without doubt that both factors exert an injurious effect, the one on the other.

A recent study by Mettier and Minot [9] sheds important light on the rôle of achlorhydria in this syndrome. Judging the rate of iron absorption by the increase of reticulocytes in the blood stream, they were able to demonstrate that if, after an initial rise due to the ingestion of iron salts, the iron was again given in an acid medium in such a way that the duodenal and jejunal juices were kept acid for a number of hours, a second reticulocyte rise was obtained. From this work it is inferred that the absorption of iron, like that of other minerals, is impaired when there is an absence of free hydrochloric acid, and that a person who shows achlorhydria is susceptible to the development of a simple anæmia which is the result of this lack of iron absorption. They refer to the work of Mitchell and Miller,[13] who found that spinach (rich in iron) was of maximum potency in regenerating the hæmoglobin of the experimental anæmia of dogs when it was given in the form of the acidulated ash. If, in addition to achlorhydria, there is a dietetic deficiency, then two factors are present—a poor intake of iron and a poor absorption of iron.

In this achlorhydric anæmia the diet is deficient in iron and there is a gastritis resulting in absence of hydrochloric acid—two factors with the same end-result—an anæmia which is quickly relieved by the administration of iron.

If a person is severely anæmic as a result of bleeding from a peptic ulcer, there is usually a rapid rise of hæmoglobin and red cells, and seldom is any hæmatinic remedy called for, although the red cells and hæmoglobin may reach a very low level, despite a rigid diet of milk and other foods poor in iron. In these patients the iron stores of the body are probably readily available, and, in addition, the gastric secretion is powerful, resulting in a optimal absorption of the available iron. Those, on the other hand, who are resistant to return to normal levels are either shown to be continuously oozing or are often shown to possess no free hydrochloric acid.

Such a failure to respond was observed in a woman who had a copious hæmatemesis the day before she came under observation. She showed a hæmoglobin percentage of 15 and a red cell count of 2·4 million. Although no further evidence of bleeding was found, the hæmoglobin did not rise above 25 per cent. for nine weeks, and it was only 45 per cent. two weeks later; she showed, however, achlorhydria with excess of mucus, although no organic cause could be found on radiographic examination. This patient had suffered from "gastritis" for years, and had complained of pallor for the previous seven years. Is the delay to return to normal blood values exemplified by this patient related to her gastric deficiency?

The rapid response of the patient following bleeding from an ulcer may therefore be the direct result of his powerful gastric secretion, while that person showing achlorhydria is already at a disadvantage with low and impoverished iron stores through a long-standing deficient absorption of iron from the food.

ANÆMIA AFTER GASTRECTOMY AND GASTRO-ENTEROSTOMY.

The presence of secondary anæmia following gastrectomy and gastro-enterostomy is well known. It appears that the incidence of anæmia in gastrectomy depends on the extent of the gastrectomy, and an interesting paper by Morley [14] has also suggested that the rate of emptying is an important factor in the development of the anæmia; the more quickly the stomach empties the more likely the development of anæmia. He has shown how the Schoemaker operation of gastrectomy, with its longer gastric retention of food, is not usually followed by manifestations of anæmia, while a Polya is frequently followed by a secondary anæmia. In addition to anæmia, typical and advanced atrophic tongue mucosal changes can be seen in patients years after

gastro-enterostomy. Gordon Taylor and colleagues [15] noted a similar atrophic mucosa of the tongue following gastrectomy. Since all the manifestations of this syndrome of achlorhydric anæmia can be seen in such surgical cases it appears unnecessary to catalogue them separately; they can be regarded as examples of the complete syndrome in which the operation of gastrectomy or gastro-enterostomy has produced the changes essential to the development of the disorder.

Seven of the present group of 55 were patients who had had a gastro-enterostomy performed many years previously, between 25 and 4 years before the manifestations of anæmia. As is seen from Table II., six showed varying degrees of anæmia, while the seventh (No. 6), although showing typical changes in the tongue and in the nails, still possessed a good

TABLE II.—*Post Gastro-enterostomy Anæmia.*

—	Sex.	Age.	Time of operation (years ago)	Red Cells.	Hæmoglobin per cent.	Tongue	Nails.
1	F.	37	16	3·0	33	Glazed.	Spoon-shaped
2	F.	42	10	4·0	55	,,	Flat and brittle.
3	M.	56	4	3·6	56	Sl. gl.	Nil.
4	F.	52	20	4·6	57	Glazed.	Flat and brittle.
5	F.	42	7	4·2	40	,,	,,
6	F.	56	25	4·9	85	,,	,,
7	F.	45	17	2·8	35	,,	Brittle.

Gastric Juice.—Achlorhydria in each case.
Sl. gl. = Slightly glazed.

hæmoglobin level. The first case showed extreme nail changes, while five complained of brittleness and flatness of the nails. No free hydrochloric was obtainable on ordinary fractional analysis, although one patient did show hydrochloric acid after the injection of histamine. Inquiry into the diet disclosed the same dietetic faults in four of the seven cases, while the remaining three were in the habit of taking some meat daily.

The response to iron administration was as good in five of these operated cases as in the "idiopathic" cases. It appears, therefore, that when an operation produces a change in gastric function, such as to result in achlorhydria and rapid emptying, there is a danger of constitutional sequelæ—anæmia, atrophy of the tongue mucosa, and brittleness of the nails. An inadequate diet would accelerate this anæmia, and iron administration would correct it, as well as improving the state of the nails and tongue mucosa.

THE PLUMMER-VINSON SYNDROME.

In considering the cause of this disorder, the syndrome first described by Paterson [16] and Brown Kelly [17] and now called the Plummer-Vinson [18] syndrome, deserves attention. In addition to the dysphagia, there is a long-standing anæmia, atrophy of the tongue, pharyngeal and œsophageal mucosa, a sallow complexion, painful cracks at the angles of the mouth, and changes in the character of the nails. Indeed, the syndrome differs only in the additional dysphagia. Study of the gastric secretion is difficult in this syndrome, but of six examined by R. D. Owen and myself four showed achlorhydria and two showed a secretion of acid after the injection of histamine. Cameron [19] and Witts [1] have also made similar observations; in some cases achlorhydria was found, but it does not seem so common a feature as in simple achlorhydric anæmia.

The laryngologists are inclined to believe that the dysphagia is the primary abnormality, but it appears that atrophic changes are always observed when the patients first complain of dysphagia, although they may state that they have suffered from a "small swallow" for years. In any case, as a result of the dysphagia the diet is a very restricted one of "slops" and very similar to that of the syndrome under discussion.

A typical example showing the course of events is the following, in a woman aged 50, who had suffered from flatulence and discomfort after food for years and who had confined her diet to starchy foods and rarely ate meat, vegetables, or fruit. Her appetite was poor and the bowels constipated. Her swallowing gave rise to occasional difficulties, and necessitated constant caution and much patience, but she had not consulted any medical practitioner for this symptom. She had recently experienced palpitations and dyspnœa, for which she sought relief. In addition to the lemon pallor, the lips showed the angular fissures, the tongue showed well-marked atrophic changes, and the nails were flat and brittle. The red cells were 3·7 million, and the hæmoglobin 40 per cent. Achlorhydria was present. As a result of iron administration she felt greatly improved, and the blood showed a return almost to normal—red cells 4·6 million and hæmoglobin 80 per cent.

Remarking on the effect of the passage of an œsophageal bougie, Paterson [20] recently stated: "Increased ability to swallow immediately follows the passage of a tube, and if this be reinforced by assuring the patient that she is now able to eat solid food, an improvement in the general condition is soon apparent. With the rise in the hæmoglobin content of the blood and the shrinking of the spleen, the relation of the anæmia to the restriction of food is emphasised."

A still closer relationship between these two types of anæmia is exemplified by the following case:—

A woman, aged 42, had enjoyed comparatively good health until seven years ago when, for stomach pains and evidence of an ulcer, a gastro-enterostomy was performed. After the operation digestive symptoms reappeared and the appetite failed. For the last six years the diet has been grossly deficient and anæmia has become apparent. Twelve months ago the tongue mucosa was noted as atrophic; she had frequent cracks at the angles of the mouth, the sclerotics were bluish, and the nails were flat and brittle. The blood showed the usual features: red cells 4·2 million and hæmoglobin 40 per cent. Achlorhydria was present, with hydrochloric acid secreted after histamine.

In other words, the case was typical of simple achlorhydric anæmia. Within the last year dysphagia has appeared and has increased in severity. The condition is now that of the Plummer-Vinson syndrome and the sequence of the gastro-enterostomy, then the anæmia and the atrophy of the mucosa, and lastly the dysphagia, suggests that the primary change in the syndrome is that of the atrophy and that the dysphagia is secondary.

The effect of pregnancy on this simple anæmia is also of some interest. Attention has already been drawn to the exacerbations, the patient obtaining some symptomatic relief with the termination of pregnancy. It is believed that the offspring obtains most if not all of its iron through the placenta, and since the iron content of the liver and other solid organs is high at birth it is reasonable to conclude that pregnancy results in a great depletion of the maternal iron stores, and in consequence an exacerbation of the anæmia in those whose stores are low. Recently Strauss [21] described three cases of this syndrome in women who were pregnant, and in whom the anæmia had become apparent during the pregnancy. In the three the effect of iron therapy was striking.

Metabolism studies on iron in this disorder are lacking, although there is uniformity in the reports of the beneficial effect of iron. As long ago as 1895 Kunkel [22] showed that the liver, spleen, and blood of animals contained an excess of iron after the administration of iron salts with the food. It is now believed that iron, in whichever form it is given, can be demonstrated in increased amounts in such organs as the liver. Recent estimations of the iron in the serum [23] (as distinct from that contained in the

hæmoglobin) have shown that, while in pernicious anæmia there is an increase in this value, in some cases of long-standing secondary anæmia as great a decrease is found as in animals rendered anæmic by repeated blood loss. When iron was given to such persons the improvement noted clinically corresponded with an increase in the serum iron. It must, however, be stated that such low values were obtained only seldom, and many cases of secondary anæmia examined by Riecker showed no fall in the serum iron. From these observations he concludes that medicinal iron is seldom required.

The important ætiological features, then, are the deficient diet, poor in iron, the achlorhydria with its impaired absorption of iron, and the simple anæmia with its deficient hæmoglobin and its therapeutic response to iron. All these support the conception of a primary iron deficiency as the essential cause. That such a syndrome develops after gastro-enterostomy (many years after operation) is also in keeping with the hypothesis of an iron deficiency. That a similar picture is also seen in the Plummer-Vinson syndrome further supports the conception of an iron deficiency. I suggest that the first change to occur is the "gastritis," a slow and insidious process, possibly the effect of a blood-borne infection; the result of the "gastritis" is seen in the symptoms of indigestion, and gradually the diet becomes more and more restricted to carbohydrate foods; the iron of the body becomes gradually depleted, and through both factors —a deficient iron intake and a defective iron absorption—this depletion is further enhanced; the production of hæmoglobin fails and anæmia becomes more a prominent feature of the syndrome. When iron is given the anæmia improves.

The Importance of the Disease.

The condition is of importance not only on account of its frequency in middle-aged women, causing a chronic state of anæmia and invalidism, but also because of the widespread nutritional changes that are seen in the fully developed syndrome. Indeed, it appears that the secondary anæmia, the atrophic changes in the tongue mucosa, and the abnormal brittleness of the nails are all manifestations of one and the same deficiency; for not only does the anæmia improve on the administration of iron but, in addition, the tongue mucosa becomes more normal and the nails less brittle and (if previously mal-shaped) grow normally.

The nail changes have only been observed in this disorder and the allied malady, the Plummer-Vinson syndrome. They are not encountered in advanced anæmia and in pernicious anæmia, the nails are always normal to all appearances, so that anæmia per se does not materially alter them. But, as has already been suggested, it is highly probable that in both conditions, simple achlorhydric anæmia and Plummer-Vinson syndrome, an iron deficiency is present. Furthermore, since improvement in the character and shape of the nails follows the administration of iron it can be concluded that the bad condition of the nails is intimately related to a long-standing iron deficiency.

Atrophy of the tongue mucosa is seen in many diverse conditions. It is often well marked in pernicious anæmia, subacute combined degeneration of the cord without anæmia, sprue, ulcerative colitis, cœliac rickets, intestinal strictures, and many other alimentary lesions. I have recently observed two cases with repeated intestinal hæmorrhage over a period of three months, and both have developed a glazed tongue while under observation. That the tongue mucosa shows an improvement after liver or stomach extract has been noted by several observers,[24 25] and Keefer[26] shows photographs of the improvement in cases of anæmia following upon correction of the malnutrition. Similar improvement in the tongue mucosa after iron can be observed in the patients with simple achlorhydric anæmia. Although no actual regeneration of papillæ can be noted, the tongue ceases to be bald and smooth, and becomes less red. The almost constant changes in the tongue in this syndrome and the absence of any history of soreness of the tongue are suggestive of a nutritional disorder, and again may be related to an iron deficiency.

If, therefore, these changes of cutaneous and epithelial structures are nutritional in origin and eventually prove to be an indication of an iron deficiency, then further functions must be attributed to iron in the maintenance of general nutrition.

The relationship between the genesis of this simple anæmia and surgery of the stomach is also of some importance. Such a complication cannot be common, although it probably occurs more frequently than has been realised. That achlorhydria is essential to the development of the syndrome in these operated cases seems also true and, since Lindsay and Evans[27] showed that in a series following gastro-enterostomy only 18 per cent. had persistent achlorhydria, it can be concluded that only a minority possess this disadvantageous state of achlorhydria and therefore become liable to a secondary anæmia. With this achlorhydria, coupled as it is with a rapid emptying, there is a danger of a defective absorption of iron from the food, and when this is further associated with a diet poor in iron, then a chronic anæmia is most likely to develop. It is sometimes difficult to regard the secretion of hydrochloric acid as a valuable asset when one is confronted with the complications of gastric ulcer, but hydrochloric acid is a normal secretion and, when any mechanism such as gastro-enterostomy results in its suppression, the result may be chronic anæmia.

Relationship to Pernicious Anæmia.

For some years it was thought that the gastric defect played little part in the development of pernicious anæmia, but recent work has shown that the gastric achylia is the real and essential basic lesion. It might be asked, since achlorhydria is found in both types of anæmia, why one person should develop a simple secondary anæmia and another a primary anæmia; for in both the achlorhydria has been in existence for years. There are, however, certain differences.

In the primary type of anæmia the customary history is that the patient, although troubled with a certain degree of flatulence, has been able to digest normal food, and that he has only suffered from anorexia with the advent of the anæmia. Again, examination of the gastric secretion shows the absence of both acid and pepsin; as a result of this atrophic function there is eventually a deficiency in the supply of the necessary factor for the normal maturation of the red cells, with its consequence—a megalocyte anæmia. In spite of the absence of hydrochloric acid, the diet has been so varied throughout the years before the onset of anæmia (Cornell[28]) that adequate absorption of iron has taken place, and, in consequence, although a failure in red-cell production may occur at any time, sufficient iron is available for hæmoglobin production, and the colour-index remains above unity.

This contrasts with the state of affairs in the simple type of anæmia, where the diet has been abnormal for years. The intake of iron has been deficient and, through the absence of hydrochloric acid, the absorption of the little iron present has been defective.

The continued secretion of pepsin and the appearance of acid in some of the patients on stimulation with histamine suggest that the gastric lesion is not of the type which leads to a deficiency, as seen in pernicious anæmia—a deficiency in red-cell production. With the long-standing defective diet and the poor absorption of iron, failure of hæmoglobin production becomes the essential feature, while the red cells are more or less unaffected. That achlorhydria does not of necessity result in an iron deficiency is shown not only by patients with pernicious anæmia (in whom there is always sufficient iron available), but also by more or less normal people who show no anæmia, in spite of achlorhydria. These people generally complain little of indigestion and enjoy a normal diet and, in consequence of this normal diet, presumably are able to ingest sufficient iron for their requirements.

Since both types of anæmia are so intimately related to gastric function, it would not be surprising to meet an intermediate type possessing characteristics of both a primary and also a simple anæmia. Such cases are not common. Six individuals of the present group showed a complete achylia—a secretion identical with that encountered in pernicious anæmia.

A female, aged 48, with a moderate degree of anæmia had 3·9 million red cells and 44 per cent. of hæmoglobin. She showed a good response to iron in spite of the achylia. Time only will prove whether, with such a secretion, the patient will show characteristics of a primary anæmia and will need liver in addition to iron to remain well. It is probable that years must elapse before such a deficiency is made obvious, for it is known that an abnormal gastric secretion has been found 15–20 years before the development of any manifestations of anæmia.

Another who showed achylia was a man aged 58; when first seen in 1929 he showed a severe degree of secondary anæmia—red cells 2·6 million, hæmoglobin 26 per cent. The tongue surface was atrophic, the spleen was palpable, and the nails were normal. As a result of liver extract his blood count rose in nine weeks to: red cells 4·5 million and hæmoglobin 80 per cent. He took no further liver for a period of eight months, when he developed an equally severe grade of secondary anæmia, which again responded to liver extract.

It appears that if an anæmia, although of low colour-index, is characterised by a big reduction in the red cells and also macrocytosis in the blood films, and this be associated with a true achylia, the administration of both liver and iron may produce a better response than either would alone. Such an anæmia is far less common than the ones "true to type."

In Table III. the chief differentiating features between the two types of anæmia are shown.

TABLE III.

	Simple achlorhydric anæmia.	Pernicious anæmia.
Duration of anæmia	Indefinite; 5–10 years or more.	Two years max.
Gastro-intestinal symptoms ..	Usually marked and long-standing.	Not prominent.
Diet	Abnormal for years.	Normal.
Tongue mucosa ..	Atrophic changes constant.	Atrophic changes less frequent.
Nails	Changes common.	None.
Neural signs ..	Uncommon.	Common.
Bilirubinæmia ..	Decreased.	Increased.
Gastric secretion ..	Achlorhydria.	Achylia.
Red cells	Not greatly reduced; microcytes.	Marked reduction; macrocytes.
Hæmoglobin ..	Relatively low.	Relatively high.

Treatment.

"But even if the assimulation of inorganic iron was a proved fact, it would have no importance in medical practice, since, as our experiments show, the iron required for the formation of hæmoglobin is much more readily and plentifully assimilated from the organic iron compounds of our normal dietary. Hence there is in no case any reason to prescribe preparations of iron for the production of hæmoglobin in people who can take their natural food with a good appetite."

Thus did Bunge[39] state his views on the therapeutics of iron in 1889. The further observations of clinical experience have not resulted in any essential alteration in this statement, and, in fact, it is peculiarly apt when this disorder and its response to treatment are considered. For, as has already been emphasised, the food partaken of by these patients is far from being normal or natural, neither do they possess a good appetite. In consequence, if it is agreed that there is an iron deficiency in this disease, material improvement following iron administration should be expected, and upon this there is general agreement, provided iron is given in sufficiently large doses. Satisfactory results have been published by Kaznelson,[10] Meulengracht,[30] Witts,[31] Schulter,[32] and others.

I used ferri et ammon. cit. in doses of grs. 60–80 a day. Provided this is taken over a period of 6 to 12 weeks, there is a decided rise in the hæmoglobin; indeed, the clinical improvement is as striking as that observed in pernicious anæmia with stomach or liver extract. After many years of chronic anæmia the return of a normal hæmoglobin content is accompanied by a well-being which is very new to these invalids.

Of the present series of cases some were only seen once, to obtain treatment elsewhere, some were given additional hæmatinic remedies, others were careless and did not persevere with treatment, while the remainder—those that faithfully ingested the iron, as prescribed, over a sufficiently long period—invariably showed a rise in the hæmoglobin as well as a striking improvement in the general health. Table IV. gives the red-cell and hæmoglobin values before and after iron administration in patients who

TABLE IV.—*Response to Iron.*

Case.	Initial blood count.		End-result.		Iron Med.†	Case.	Initial blood count.		End-result.		Iron Med.†
	R.C. mill.*	Hb. %	R.C. mill.*	Hb. %			R.C. mill.*	Hb. %	R.C. mill.*	Hb. %	
1	3·3	30	5·2	95	2 m.	28	4·6	57	5·0	95	2 m.
3	4·05	55	5·0	96	2½ m.	29	4·3	46	5·2	82	3 m.
4	5·0	52	5·75	85	6 w.	30	3·8	25	4·8	84	3 m.
5	4·8	48	5·0	98	3 m.	31	3·7	40	4·6	80	3 m.
17	2·8	35	4·9	75	3½ m.	32	4·7	53	5·0	92	2½ m.
18	3·8	48	5·2	86	2½ m.	33	2·4	40	4·0	85	2½ m.
20	4·6	65	4·8	110	2½ m.	34	4·3	30	4·5	82	4 m.
21	3·76	46	5·4	70	6 w.	35	4·7	38	4·8	84	3 m.
23	2·5	25	4·6	75	3 m.	36	2·5	22	4·5	70	5 m.
26	4·1	42	4·9	85	2½ m.	52	3·1	34	4·3	78	3 m.

* R.C. mill. = red cells in millions. M. = months; w. = weeks.
† Iron med. = duration of iron medication.

were not given any other form of treatment. An improvement in the hæmoglobin was obtained in all, but as the blood estimations were not done regularly "the duration of treatment" is a somewhat arbitrary figure.

No untoward symptoms have been noted as a result of the ingestion of these large doses of iron, and only one patient was unable to tolerate grs. 25 three times a day. It is not known why such large doses are necessary, and little is known of the fate of the iron that is absorbed, other than its storage in the liver. It is necessary to continue with smaller doses of iron to prevent relapse, as was shown by some of this series in whom the hæmoglobin fell to its original level of 40 per cent. or so in six months on the

cessation of iron therapy. When once the hæmoglobin has reached the 95–100 per cent. level the dose was reduced to gr. 10 three times a day.

In addition to the improvement in the anæmia, the nails and tongue changes become less marked, and in some patients the original departures from normal have completely disappeared, although no actual regeneration of papillæ has been observed in the tongue mucosa. Furthermore, some patients show a gain in weight following general improvement; one patient increased in weight from 6 st. 8 lb. to 7 st. 8½ lb. in nine weeks.

It is a question whether iron should be prescribed to the individuals who show achlorhydria, atrophic changes in the tongue, and undue brittleness of the nails without any marked reduction in hæmoglobin. Several such patients have been given iron and, although exact proof of benefit is difficult to record when the hæmoglobin is already above 80 per cent., yet the return of well-being and vigour has been sufficiently marked to warrant iron administration even in these cases.

The treatment of the digestive disturbances is a more difficult project. It is probable that the anæmia would gradually disappear if the gastric secretion could be re-established, and, as a result of this, appetite and hunger for meat and other articles rich in iron could return to the patient. To attain this, washing out the stomach, to free it of its thick coat of mucus, would be a direct benefit, as Hurst [33] has emphasised. Two patients, previously showing achlorhydria on ordinary fractional analysis, showed a return of acid on a similar examination three and four weeks after daily lavage of the stomach; but as the rise in hæmoglobin was too slow, and domestic reasons forbade delay, these patients were later given iron in addition. Keefer [34] has recently shown that an improvement in an existing anæmia results in a better gastric secretion, and that some of his cases of malnutrition anæmia (tropical in origin), previously showing achlorhydria, showed a return of free acid when their anæmia was corrected. He concludes that achlorhydria is often the result of the anæmia. Whilst it must be readily admitted that such improvement is conceivable and natural, it would be difficult to regard the anæmia in this syndrome as the causative factor in the development of achlorhydria, for several patients with little anæmia yet show achlorhydria, and all the factors point to the gastric abnormality as the primary change. Six of the patients who showed a good response to iron alone were re-examined after they had returned to normal figures, but no improvement was noted in the gastric secretion.

The administration of hydrochloric acid has been frequently tried and applauded, but it is extremely difficult to subsidise the natural secretion. Many years ago Zander [35] claimed that hydrochloric acid administered to a patient with chlorosis was of more lasting benefit than iron, for, in his opinion, it caused a better absorption of iron from the food. Stockman [36] and others failed to confirm these observations in chlorosis. The position is much the same to-day, and the concensus of opinion seems to be that hydrochloric acid administered to a patient with achlorhydria cannot be of much therapeutic importance except in post-prandial diarrhœa. However, there are some patients in whom flatulence and discomfort after food is especially relieved by either hydrochloric acid alone or a mixture containing hydrochloric acid and pepsin. I have frequently obtained symptomatic benefit in this type of patient from a mixture containing a drachm of hydrochloric acid and a drachm of glycerin of pepsin, made up with orange or lemon water, taken after or during meals. Some patients seem to derive no benefit from hydrochloric acid; in fact it appears to increase the discomfort. Others find relief from a morning dose of alkali powder taken half an hour before breakfast; this probably acts by removing the thick coat of mucus and allowing a better gastric secretion during the meal.

Conclusion.

Simple achlorhydric anæmia appears to be a common and chronic anæmia of adult womanhood. The evidence available is suggestive of an iron deficiency, consequent on a defective diet, and an impaired gastric secretion. The disorder can be therapeutically corrected by the administration of iron, but this does not remove the cause. Attention to the gastric secretion and measures to improve it should ultimately result in a better intake and a better absorption of iron. The disease is worthy of attention, not only on account of its frequency in women and the ease of its correction from the standpoint of the anæmia, but also on account of its appearance in patients who have undergone surgical treatment to the stomach. Its development is a further index of the normal function of hydrochloric acid secretion. In addition, it is possible that the malnutritional changes, such as those in the tongue mucosa and entrance to the food passage and in the nails, are related to a long-standing deficiency of iron.

It is a pleasure to acknowledge the kindness of the honorary physicians of the Middlesex and Royal Free Hospitals in allowing free access to the patients under their care.

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CONGENITAL ELEVATION OF THE DIAPHRAGM,

WITH REPORTS ON SIX CASES.

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Congenital elevation of the diaphragm is a condition due to faulty development of this organ, as the result of which the diaphragmatic dome on one side, usually the left, forms a sac bulging upwards into the thorax and containing displaced abdominal viscera.

The condition was first recognised and described by Jean Louis Petit (1674–1750), who gave it the name of *eventratio diaphragmatica*. His collected writings, however, were not printed until nearly a quarter of a century after his death, when his pupil, Lesne, published them as a memorial of a master greatly beloved.[1] It is thus to Lesne that we owe the preservation of Petit's account of the first known case. If medical precedent had been followed and the condition were known as Petit's disease, fitting tribute would have been paid to one whose name should not be allowed to pass into oblivion.

After describing diaphragmatic hernia, Petit records another example of diaphragmatic displacement which, in his opinion, must have been due to an error in development. The patient had suffered for long from attacks of asthma, aggravated by eating, and had died after an attack of "inflammation of the stomach." At autopsy Petit discovered that the left side of the diaphragm projected upwards into the thorax, forming a large sac. The base of the left lung was glued to this by adhesions, while, on opening the belly, the sac was found to contain the stomach, colon, and mesentery, matted together as the result of recent inflammation. The diaphragm was intact, and Petit lays stress on the fact that there was no rupture in its vault nor any separation of its muscular or tendinous fibres. The case thus differed from cases previously described, in which he had noted a true hernia of the diaphragm.

Cases were subsequently recorded by Froriep[2] (1837), Lawrence[3] (1852), Howard Marsh[4] (1867), Thoma[5] (1882), Tennant[6] (1894), Kronig[7] (1898), and others in succeeding years. It is to Woodburn Morison, however, that we owe the first complete account of the condition, and his admirable monograph[8] on the subject has already taken its place among the classics of medical literature. Each of the cases of diaphragmatic eventration referred to above was discovered at autopsy, and the descriptions refer to appearances found post mortem.

The first instance of the application of X rays to the diagnosis of diaphragmatic eventration that we have been able to discover in medical literature was made in 1901 by Widenmann.[9] The patient, a man aged 48 years, who had suffered from repeated attacks of pain in the chest, was X rayed. The skiagram revealed a dome-shaped shadow projecting upwards into the left side of the thorax. Proof that this shadow was cast by the diaphragm, and that the stomach lay immediately beneath it, was forthcoming when a sound of zinc and small shot was passed into the stomach by way of the œsophagus, and could be seen curling upwards in the clear area immediately below the diaphragmatic arc.

A further application of modern diagnostic methods to the examination of the malformation was made by Baetge (1913),[10] who utilised the electro cardiogram. He claimed that the tracings showed a departure from the normal owing to the displacement of the heart to the right. We have, however, been unable to confirm this, for electro cardiograms of two of our cases, kindly taken by Dr. A. J. Scott Pinchin, showed that the wave forms were not abnormal.

A still more ingenious method of differentiating between gastric hernia and eventration was successfully employed by Hildebrand and Hess.[11] A tube passed into the stomach communicated with a manometer which enabled the intra-gastric pressure to be estimated during the two respiratory phases. Obviously, if the stomach is in the abdominal cavity, intra-gastric pressure will rise during inspiration and fall during expiration, but if the stomach is situated in the thoracic cavity—i.e., if there is hernia of the diaphragm, the reverse effect will be noted.

Ætiology.

The diaphragm is raised in many pathological conditions: (1) Certain abdominal conditions—e.g., ascites, pregnancy, subphrenic abscess. (2) Pulmonary diseases—e.g., tuberculosis, especially of the fibroid type, fibroid lung, atelectasis, neoplasm. (3) Paralysis of the phrenic nerve due to disease, trauma, or operation. (4) Temporary elevation of doubtful origin may occur in children or in adults as the result of trauma. An example of the latter is shown by the following case:

A man, aged 58, whilst working, hit the left side of his chest against an iron grating. Considerable bruising and swelling resulted, and a fracture of the lower ribs was suspected. An X ray photograph was taken but no fracture was seen. On the other hand, the left dome of the diaphragm was found to be raised to the level of about the ninth rib behind. Two months later a second film was taken. In this radiogram both domes of the diaphragm were at the usual level. All pain and swelling had disappeared and the patient stated that he was quite well. The elevation of the left dome of the diaphragm in this case thus appears to be related to the injury to the chest wall, and might be explained as a reflex phenomenon designed to prevent the pain that would have resulted from a contraction of the diaphragm on the injured side.

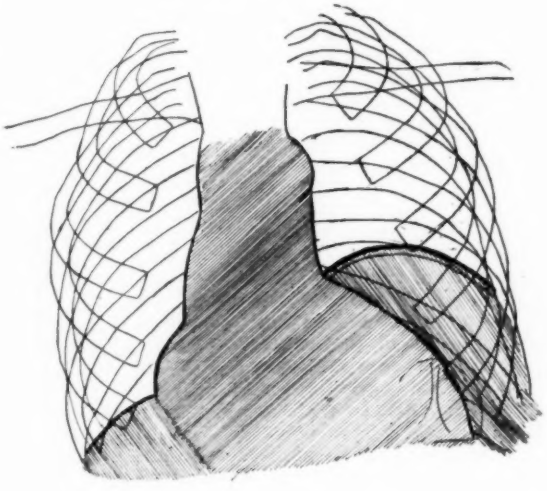
In all these cases the cause of the rise in the diaphragm is obvious. It is characteristic of eventration that a considerable degree of elevation is present in the absence of any demonstrable cause. There is no increase in intra-abdominal pressure, there is no disease of the lungs, and in at least one case in which a microscopical examination of the phrenic nerve was made after death, the sections showed no abnormality,[12] and though, in another case, Benda found reduction in the size of a phrenic nerve, the fibres were normal.

There seems, indeed, no reason to doubt that Petit was correct in regarding the condition as a congenital defect, for it has been found at the autopsy on a child who only lived 24 hours, and even in a fœtus. It is worthy of note, also, that in nine of the reported cases elevation of the diaphragm was accompanied by other congenital abnormalities.

If eventration of the diaphragm is a congenital condition the development of the diaphragm is worthy of consideration. The pericardial, pleural, and peritoneal cavities of the adult are formed by subdivisions of the embryonic body cavity. The anterior end of the cœlom containing the heart is shut off by a partition which forms at about the sixth week of intra-uterine life and, at a slightly later date,

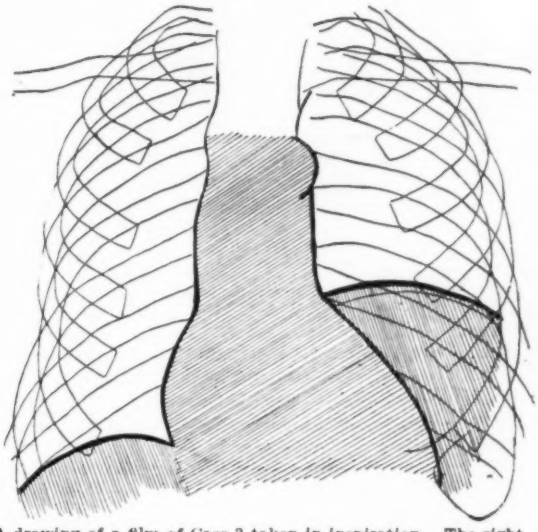
the two pleural sacs are separated from the peritoneal cavity by the completion of the diaphragm. The greater part of the latter is formed from the septum transversum, which is ventral in situation, while the dorsolateral portions are derived from the pleuro-peritoneal membranes which close the communicating ducts between the two cavities on the right and left sides. The musculature of the diaphragm is stated to be developed from two muscle masses, derived from the fourth cervical myotome.[13] These grow into the developing diaphragm while the septum transversum is still in the cervical region. Later the diaphragm migrates caudally to reach its proper place.

FIG. 1 (Case 3).



A drawing of a film taken in expiration to contrast with one in inspiration. The right dome of the diaphragm is at the level of the upper margin of the anterior extremity of the fifth rib, and the left dome at the level of the anterior extremity of the third rib.

FIG. 2 (Case 3).



A drawing of a film of Case 3 taken in inspiration. The right dome of the diaphragm is now opposite the anterior extremity of the sixth rib, whilst the left dome, which is flatter than in the previous film, has moved down to the anterior extremity of the fourth rib. A definite contraction of the left dome of the diaphragm has thus occurred.

In congenital elevation the right side of the diaphragm is in its normal position, whilst the left cupola forms a thin sac with normal attachments, but projecting into the thorax to reach a high level. It seems, probable, therefore, that neither a failure of migration nor a failure of union of the component parts (a cause of hernia of the diaphragm) is responsible for the condition, but a failure of the development of the musculature on one side. As a result of this, the muscle sheet is almost entirely replaced by a thin membrane, which yields to abdominal pressure. Such a sequence might be expected, for the pressure in the pleural is lower than that in the abdominal cavity, and a membranous diaphragm would fail to fulfil its pressor function.

Clinical Aspect.

The malformation is generally discovered by chance in the living as the result of radiological examination or operation, or in the dead at autopsy. There are no distinctive symptoms, though dyspnœa and dyspepsia have often been noted.

The time-honoured methods of inspection, palpation, auscultation, and percussion, though we cannot afford to dispense with them, have only a limited value in the investigation of obscure disease of the chest. The excellent quality of modern radiograms of the thorax has compelled even the conservative to admit that a skiagram forms an essential part of a complete examination of the chest. In the opinion of the more progressive, an X ray examination is the essential part. It is true that a good physical examination is better than a poor skiagram, but a good skiagram furnishes more reliable information than that obtained by any other method. The application of the classical method of physical examination in one of our cases (Case 1) led to an entirely erroneous conclusion. The patient complained of dyspnœa, there was a family history of tuberculosis, the percussion note was impaired at the base of the left lung, vocal fremitus could not be felt, and vocal resonance was inaudible over the dull area. There was also displacement of the heart away from the affected side. A tentative diagnosis of tuberculous pleural effusion seemed justified. A skiagram demonstrated an elevation of the diaphragm, and a single glance at the film disclosed more than a careful clinical examination had been able to discover. The quickest method of revealing the secrets of the thorax, like those of Joanna Southcote's box, is the intelligent application of the X rays. A brief description of the physical signs will therefore suffice.

On inspection there may be diminished movement of the affected side of the chest—almost always the left side. The presence of Litten's sign has been noted. On palpation vocal fremitus is reduced at the left base. The results of percussion will depend upon the relative amounts of fluid and air in the stomach, a dull note being elicited over fluid and a tympanitic note over the inflated part of the organ. Tympany may extend as high as the fourth rib anteriorly. On auscultation breath sounds and vocal resonance are absent over an area approximately corresponding with the height of the diaphragm. A succussion splash is usually evident, for the stomach is rarely empty.

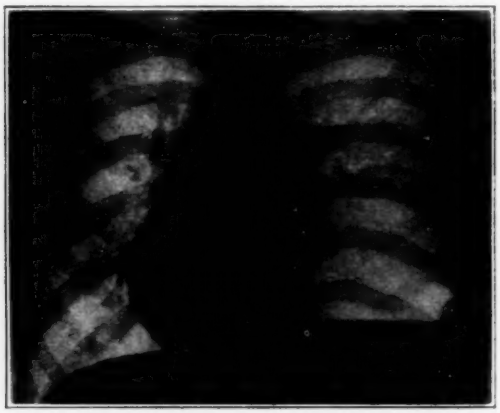
RADIOLOGICAL EXAMINATION.

The following descriptions refer to left-sided eventration.

Fluoroscopy shows the diaphragmatic movement.

During quiet respiration the unaffected dome exhibits normal excursions. These are usually well marked, but there is no evidence of exaggeration to compensate for the absence of movement on the elevated side.

FIG. 3 (Case 1).

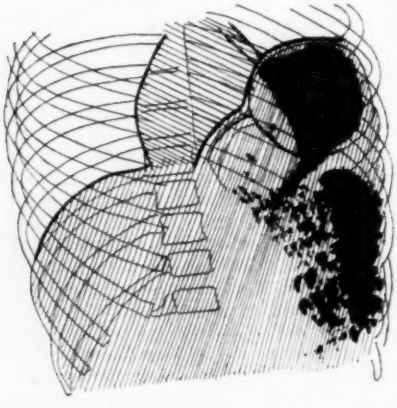


An erect anterior view taken after an ordinary meal without barium. The raised bowline of the left dome of the diaphragm and displacement of the heart to the right are shown. In addition three fluid levels are seen in the stomach, disposed one above the other.

When the patient is instructed to breathe deeply, the abnormally elevated dome will be seen to move slightly up and down, its excursions being synchronous with those of the healthy side. Only one of our cases (No. 5) showed a contrary or paradoxical movement, such as is seen in paralysis of the diaphragm. In Case 3 true contraction of the diaphragm was proved by radiograms taken in inspiration and expiration (Figs. 1 and 2).

A *radiogram* taken when the patient is recumbent shows the right side of the diaphragm in its normal situation while the left side exhibits a boldly outlined and smoothly rounded dome, the summit of which may be at the level of the second or third intercostal space. No stomach bubble can be made out in this position, but the splenic flexure, with its contained gas, is seen lying high up under the raised diaphragm. The heart's shadow is displaced to the right, and just above it the translucency of the bifurcation of the trachea is similarly displaced. Below the affected diaphragm a uniform opacity obscures the view, except in those cases in which, as noted above, some evidence of a displaced splenic flexure is found.

FIG. 5 (Case 6).



A drawing of a film taken prone ten minutes after the ingestion of a barium meal. The patient is rotated slightly into the right oblique position. The left dome of the diaphragm does not form an uninterrupted bowline as seen in the anterior view, but presents a bulge where the fundus of the stomach presses on the diaphragm. Barium is seen in the stomach and some barium has passed out into the smaller intestine.

When a radiogram of the patient in the erect posture is taken a very characteristic picture is obtained (Fig. 3). Where previously an opacity was observed in the left lower zone a large gastric air-bubble is now revealed above which arches the bow line of the diaphragm. The contour of this line is unbroken in antero-posterior and lateral views, but an oblique view may show a bulge (with convexity upwards) where the gastric fundus presses against the concavity of the thin and yielding diaphragm (See Fig. 2). Below the area of translucency, especially if the patient has recently enjoyed a good meal, can be seen two or more typically three-fluid levels in the stomach. If the surfaces of the fluid levels be traced to the left they will be seen to merge into the shadowy outlines of the splenic flexure with its usual gaseous content.

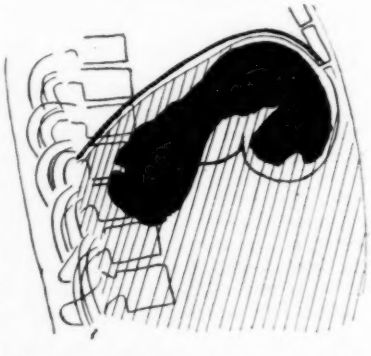
FIG. 4 (Case 6).

A drawing of a film taken in the prone position five minutes after the administration of a barium meal. The fundus of the stomach lies high up under the raised left dome of the diaphragm. The duodenal bulb is outlined by barium and points directly downwards. The second and third parts form a straight line instead of the usual loop.

In the erect lateral position the three fluid levels corresponding to those seen in the antero-posterior position may also be seen, but whereas in the antero-posterior position they are placed more or less vertically above each other, in the lateral position they exhibit a staircase formation, the stairs ascending from left to right.

Further information and confirmation of diagnosis can be obtained by examination after a barium meal. If the patient attends fasting, unless he is an air-swallower, the appearances mentioned above will not be well seen, for the gastric air bubble will be inconspicuous and the fluid levels indistinct or absent.

FIG. 6 (Case 6).



Drawing of a lateral prone view taken after ingestion of barium. The greater curvature of the stomach is displaced upwards, and together with the fundus follows the line of the raised diaphragm in a postero-anterior direction. The shape of the stomach has a resemblance to a bagpipe.

Barium meal examination.—The patient is placed behind the screen and rotated into the right oblique position. The barium is now administered and its progress down the œsophagus is watched. The cardiac orifice appears to be low in position in relation to the rest of the stomach. As the result of

this, the fluid on entering the stomach appears to pass upwards towards the fundus and then to pass downwards towards the pyloric end. The whole of the stomach is seen to be situated more to the left than usual, whilst the duodenum is also displaced to the left with its bulb pointing transversely to the right, or downwards towards the pelvis (Fig. 4). It is thus obvious that the shape and position of the stomach are abnormal. Examination with the patient lying on the back is necessary if diaphragmatic hernia is to be excluded. In this position the smooth contours of the fundus are outlined by barium, and it will be noted that none of the barium lies above the diaphragmatic level. In cases of hernia, recumbency will cause some of the barium to pass into a pouch above the diaphragm.

The appearances are equally abnormal in the lateral view. A picture taken with the patient lying down shows the smooth, curved contour of the raised dome of the diaphragm and immediately beneath it the stomach, which is distorted so as to resemble a bagpipe in shape (Figs. 5 and 6).

We were doubtful of the correct interpretation of the three fluid levels seen in the antero-posterior and lateral erect views until we were so fortunate as to secure a radiogram from the lateral aspect immediately after the patient had swallowed some barium. When the picture was taken a film of barium was still adherent to the gastric mucous membrane and the posterior wall of the stomach was clearly delineated. A curious folding of the walls was seen to be responsible for three little pools of barium: an upper one near the œsophageal opening, a smaller one just in front of this, and a larger one more anteriorly placed, the walls of which seemed to be formed by the main body of the stomach. The lateral view thus presented the appearance of a cascade with a stream of barium connecting the three pools (Figs. 7 and 8). In Case 3 an even more complex appearance was shown in the anterior and lateral

FIG. 7 (Case 4).

FIG. 8 (Case 4).

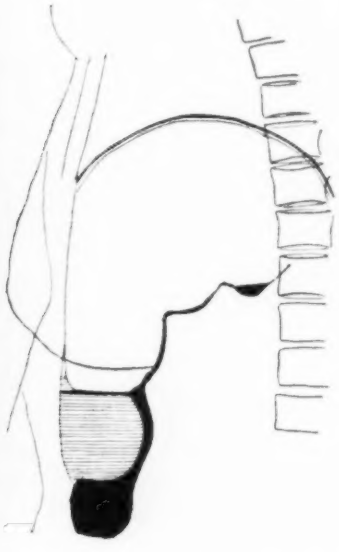


Fig. 7 is a lateral erect view of Case 4, taken immediately after some barium emulsion had been swallowed. The barium can be seen running down the posterior wall of the stomach and outlining three folds: (1) an upper posterior one which contains a small pool of barium, (2) a middle smaller fold, and (3) an anterior fold which runs down into the main body of the stomach, where there is a large fluid level.
Fig. 8 is an outline drawing of the barium meal appearances seen in Fig. 7. Compare with Fig. 10, where the arrangement was more complex.

FIG. 9 (Case 3).

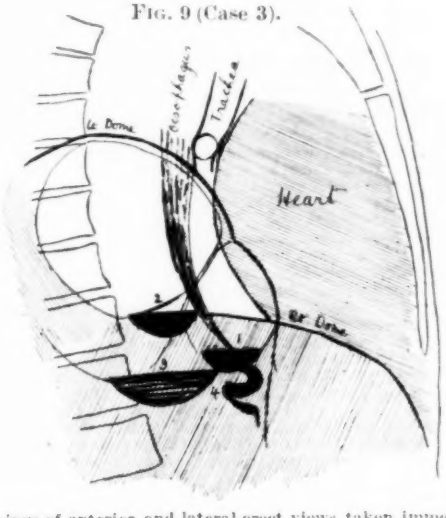
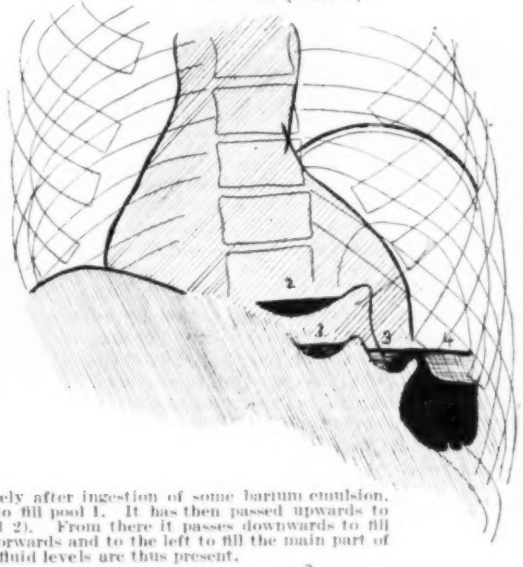


FIG. 10 (Case 3).



Drawings of anterior and lateral erect views taken immediately after ingestion of some barium emulsion. The barium can be seen running down in the œsophagus to fill pool 1. It has then passed upwards to fill a pool situated higher up and more posteriorly (pool 2). From there it passes downwards to fill pool 3, and from here a stream of barium is seen passing forwards and to the left to fill the main part of the stomach, which is situated at level 4. Four distinct fluid levels are thus present.

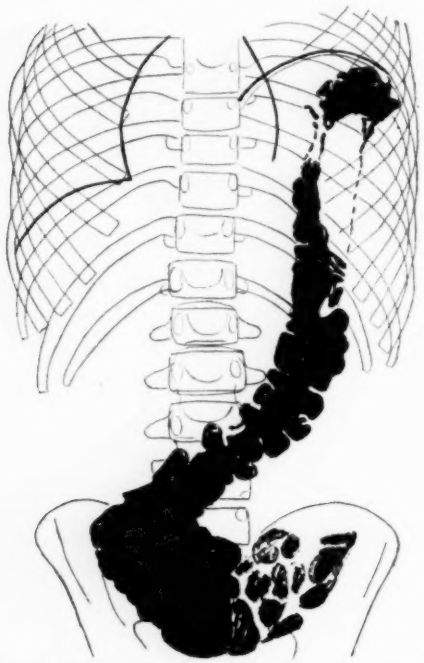
erect views, the barium outlining four possible fluid levels (Figs. 9 and 10).

If the meal is followed until it reaches the large bowel, the cæcum will probably be found in the pelvis, the hepatic flexure low in position, and the transverse colon running upwards to the left in an oblique line to meet the splenic flexure high under the raised dome of the diaphragm (Fig. 11).

Post-mortem Appearances.

Though six patients affected by eventration have attended our clinic, we have had no opportunity of comparing the results of physical and radiological examination with post-mortem findings. We have

FIG. 11 (Case 6).



A film taken when lying down six hours after the ingestion of the barium meal. The large bowel is filled as far as the splenic flexure. A residue of barium is still present in the small intestine. The cæcum is situated at the brim of the pelvis, whilst the hepatic flexure is at the level of the right iliac crest. The transverse colon runs obliquely upwards and to the left towards the splenic flexure, which is very high in position. The hepatic flexure is thus abnormally low. (This was seen also in one of the other cases.)

therefore had to rely on the descriptions of others when trying to visualise the pathological condition. The reported cases agree in their accounts of the displacement of abdominal organs and the appearance of the undeveloped and raised hemi-diaphragm. Petit describes the wall of the "sac" as formed of peritoneum, diaphragm, and pleura united by adhesions. He emphasises the absence of any rupture of the wall of the sac and also draws special attention to the fact that there was "no separation of the muscular and tendinous fibres of the diaphragm." Lawrence, who discovered eventration at the autopsy on a patient who had died of pneumonia, found that the muscular tissue only extended from the inner surface of the ribs and arcuate ligaments for a distance of 6–8 lines, and noted that such fibres as were present were only perceptible by transmitted light. The remainder of the left half of the diaphragm consisted of an aponeurotic expansion merging into the central tendon. An example of a partial atrophy is supplied by Tennant, who found eventration of the diaphragm in a dissecting-room subject. The elevated diaphragm formed a pouch extending to the height of the second costal cartilage. The anterior half of the left side of the diaphragm was normal, but posteriorly it was thin and translucent. Only a few scattered muscular fibres could be found in the affected area.

Treatment.

No curative treatment is possible. Fortunately the condition seldom causes discomfort. Symptomatic treatment for any gastric disorders, should these arise, is obviously indicated. For the rest, the patient may be reassured that his abnormality is unlikely either to lessen his efficiency or to shorten his life. He enjoys the gratification of being an interesting case whose appearance at hospital will always excite a lively concern, and he suffers few of the disabilities commonly attaching to such a condition.

Case Reports.

CASE 1.—A warehouse boy, aged 16, attended hospital on Nov. 23rd, 1926, complaining of chronic shortness of breath. One week before attendance he had had a feverish attack, associated with vomiting. He had had diphtheria in 1922, and his sister had died of tuberculous meningitis.

There was impaired percussion note on the left base posteriorly, and the breath sounds, vocal resonance, and vocal fremitus were absent. The heart was slightly displaced to the right. A succussion splash was elicited at a later visit. X ray examination showed typical congenital elevation of the left side of the diaphragm.

CASE 2.—A motor driver, aged 50, attended hospital on Nov. 18th, 1930, complaining of morning cough and retching of 12 years' duration, also dyspnœa of three years' standing. Apart from these he had enjoyed good health.

There was impaired percussion note and weak breath sounds with some coarse râles at the left base posteriorly, and elsewhere signs of emphysema. The heart was slightly displaced to the right. The liver was enlarged, ? cirrhotic. The electrocardiogram showed physiological rhythm and left side relative preponderance, and the skiagram a typical left-sided elevation of the diaphragm.

CASE 3.—A retired motor driver, aged 68, attended hospital on Jan. 27th, 1931. He had been a district railway motor driver for 45 years without losing a day through ill-health till Nov. 24th, 1930, when he was admitted to the West London Hospital suffering from intestinal obstruction. At operation performed for the relief of this six or seven days later it was noticed that the left side of the diaphragm was at an abnormally high level. The patient made an uneventful recovery and went home in 23 days from admission. The X ray showed typical left-sided elevation of the diaphragm.

CASE 4.—A dressmaker, aged 21, attended hospital on Nov. 11th, 1930. The patient had previously attended Leyton Tuberculosis Dispensary as a "suspect" complaining of four weeks' cough. Her father had died of phthisis in 1919.

Breath sounds were absent at the left base posteriorly, the percussion note in the same area being of slightly higher pitch than normal. The breath sounds in the left axilla were weak. X ray examination showed a typical left-sided eventration.

CASE 5.— A piano-worker, aged 30, attended hospital on Nov. 11th, 1930, complaining of a cough which had led his doctor to suspect pulmonary tuberculosis. He had had double pneumonia in 1921, and been bed-ridden for five weeks.

The breath sounds were weak over the upper half of the right lung and absent below the angle of the scapula. No impaired resonance or alteration of vocal fremitus or resonance was found. The liver dullness extended from the fifth intercostal space to the costal margin. The skiagram showed that the right dome of the diaphragm was raised to the fifth intercostal space in the nipple line, the left dome being at the normal level: some faint mottling suggested that tuberculous foci were seen in the left mid-zone. The right costophrenic angle was obliterated. On screen examination paradoxical movement of the diaphragm was seen, a phenomenon not observed in any of our left-sided elevations.

CASE 6.—A boy, aged 12, attended hospital on Jan. 23rd, 1930, complaining of occasional "retching" not related to meals and associated with slight pain in the

upper part of the left side of the "stomach." In March, 1928, he had been thrown out of a cart into a field on his stomach and admitted to Woodford Hospital for suspected abdominal injury; he had remained in hospital two weeks. He subsequently complained of pains in the left side of the chest and retching at times, but there was no vomiting. He had had similar attacks three or four times a week up to his attendance at hospital.

The patient was a poorly nourished boy, of small size for his age. There was impairment of percussion note at the base of the left lung, extending from the angle of the scapula to the base, and also in the left axillary region and anteriorly below the level of the fourth rib. Breath sounds, vocal fremitus, and vocal resonance were absent in this area. No succussion splash was heard on shaking the patient. A skiagram showed a typical left-sided elevation of the diaphragm.

H. G. Wells has suggested that the discovery of the secret of cancer will be made, not by a philanthropist burning with zeal to relieve the sufferings of his fellows, but by a laboratory worker consumed with an eager curiosity that has no direct connexion with humanitarian motives. The condition we have been considering may at first sight seem to be one of small practical importance. The subjects of this curious deformity are few, their sufferings are negligible; we can do nothing to restore a normal balance to the organism. But the curiosities of medical practice have their value if they stimulate the inquiring attitude of mind, and the study of even the rarest abnormalities of development may ultimately assist us to a knowledge of how to prevent the congenital deformities which in their graver manifestations are the cause of serious disability or even acute suffering.

Our thanks are due to Dr. A. J. Scott Pinchin, Dr. K. Shirley Smith, Dr. T. L. Ormerod, and Dr. H. T. Howell, who kindly allowed us to make use of their cases, and also to Dr. J. V. Sparks for helpful assistance and advice.

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GLOSSO-PHARYNGEAL NEURALGIA.

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IT has for some years been known that it is possible for a patient to suffer from paroxysmal pain in the distribution of the glosso-pharyngeal nerve. Neuralgia of this nerve is in all respects the counterpart of the much commoner neuralgia of the trigeminus, presenting the same intermissions and remissions, the same brief excrutiating pains, the same precipitation of spasms by specific movements or contacts. The difference, clinically, between trigeminal and glosso-pharyngeal neuralgia lies purely in the territory in which the pain is experienced, on the one hand in the face, on the other in the throat and ear, but especially in the throat.

Weisenberg (1910), in the course of an excellent account of a lateral recess tumour in a man of 35, for the first time drew attention to the painful syndrome of the glosso-pharyngeal nerve.

In his case the pain was produced by the pressure of a tumour on the nerve, the irritative phenomena being as follows: "(the pains) were chiefly localised to the back of the tongue, or, as he expressed it, at the 'root,' and ran down the throat to his ear. He always complained of a burning feeling in his throat, and occasionally of sensations which he likened to the crawling of roaches or flies, of a constant dryness, and of a feeling like hot or ice-cold water. He had, besides, sharp, shooting, stinging pains, which started in the back of the right side of the tongue and ran to the right ear and down the right side of the throat and, when they were exceptionally severe, to the shoulder. These lasted only for a few minutes, were sometimes agonising, and nearly always caused him to turn his head and neck to the right. Eating, especially in the last year of his illness, would always cause him pain, and his wife does not remember a single meal that he ate during this time in which he did not have attacks of pain. He did not have difficulty in swallowing, and he never choked. The pains while eating were brought on not so much by the chewing as by the contact of the food with the posterior part of his mouth and by swallowing."

Weisenberg, in critical discussion of his own case, does not specifically state that spontaneous neuralgia might originate in the glosso-pharyngeal nerve. Wilfred Harris (1921) was the first to bring forward examples of this new clinical entity, the first, that is, if we set aside, as seems proper, three cases of pharyngeal neuralgia recorded by Sicard and Robineau (1920) on the ground that these authors were not certain whether the pain originated in glosso-pharyngeus, vagus, or cervical sympathetic. Further records appeared in the American literature, notably those by Doyle (1923), Adson (1924), Singleton (1926), Dandy (1927), and Stookey (1928). Purdon Martin's (1931) paper describing a case, and discussing generally the features of the disease, if we may so term a neuralgia, is the most recent addition.

The pain of spontaneous glosso-pharyngeal neuralgia is very similar to the secondary pain described by Weisenberg. It generally strikes into the throat, and particularly at the junction of the faucial pillars with the base of the tongue, and a "trigger zone" has been noted at this point in some cases. The pain shoots from the lateral wall of the pharynx into the ear, particularly on swallowing, and it has generally first appeared during that act.

The case to be described is, so far, unique not because the pain began in the ear (for this has been noted before), but because it remained there for over 30 years before it spread to the throat.

Case Record.

A. B., a Jew aged 70, was admitted to the Salford Royal Hospital on Oct. 20th, 1929, complaining of paroxysmal pain deep in the left ear. The history of the malady extended back over a period of 36 years, with long remissions; apart from this his health had been, and still remained, excellent.

He was accustomed to have two attacks of pain each year, but latterly it had returned with great regularity every four months. He had received great benefit from medicinal treatment, and it was only recently that the failure of a prescription which he had received in Berlin led him to seek surgical relief. The pain was paroxysmal in character, began inside the left ear, and he invariably pressed his finger into the auditory meatus in an attempt to demonstrate where the pain was. The pain did not affect the auricle, but radiated deep into the head and not into the throat; it spread a little in front of the ear, but never went forward into the lower jaw, cheek, gums, tongue, or temple. The spasms, so far as he was able to tell, were not excited by anything in particular; this was an unusual feature, for glosso-pharyngeal neuralgia is generally excited by swallowing. During the pains, which were of brief duration, he grimaced, turned his head to the left, and held his face in his hands.

In the belief that this was an example of trigeminal

neuralgia of unusually limited extent, alcohol injections had already been tried, and he had six months' freedom from pain after one of these. In view of later developments this relief was probably a coincidence, but it was very misleading. The return of paroxysmal pain made it evident that something more drastic must be undertaken, and in the belief that we were dealing with an unusual trigeminal neuralgia a fractional division of the sensory root was performed under local anæsthesia, helped out by a little ether whilst the actual root was being uncovered and cut. The patient made an uninterrupted recovery, but before his discharge at the end of fourteen days there were already suspicious discomforts in the ear, of the old kind, and he seemed apprehensive. His fears were well-grounded, and three months later he was admitted with pains rather worse than before. Not having quite such firm faith then, as I have now, in the permanence of relief after fractional root section, it seemed possible that the pain might have been due to some fibres destined for the auditory meatus having escaped division, presumably owing to an anomalous grouping in the root. Accordingly, again under local anæsthesia, the wound was reopened, the root rapidly exposed, and the remaining fibres cut, and, in addition, the third division was sectioned at the foramen ovale intracranially, to allow of no possible misinterpretation of the result. This operation solved the problem, for not only did the pain in the ear return in three days, but it now appeared for the first time in the left fauces. Further, the attacks were constantly precipitated by swallowing, so that he was almost unable to drink. It was now clear that we had been deceived as to the true origin of the pain, as so many clinicians have been before us, and that the glosso-pharyngeal nerve would have to be resected if we were to cure him.

We had a choice of the posterior fossa route advised by Adson and Dandy, or a high extracranial division. In view of his age, the latter seemed to be the wiser course, though there was some risk of failure, for unless the nerve was pulled out of the jugular foramen after dissection close to the base of the skull the tympanic branch, which had caused so much trouble, would escape. Through a long incision at the anterior border of the sterno-mastoid muscle on to the mastoid process the glosso-pharyngeal nerve was sought. A free section of the fibres of the sterno-mastoid was necessary, as the man had a short, obese neck. After a very difficult dissection the glosso-pharyngeal nerve was identified high up, separated of its extracranial vagal connexions, and avulsed at the jugular foramen by strong, steady traction.

From the time of his recovery from the anæsthetic to the present day the patient has been entirely free from pain. Examined in June, 1931, fifteen months after the last operation, he declares himself to be extremely well, pain free for the first long time in 38 years, and is very active for his 71 years.

The accurate testing of sensation in the throat is extremely difficult, but the patient is quite aware of a difference between the two sides. The vagus was slightly contused during the avulsion, for he had a left side paralysis of the vocal cord and some difficulty in swallowing. This recovered slowly, but it was five months before all these additional troubles had subsided. Fortunately, this handicap impeded his recovery very little indeed. There is profound anæsthesia of the cheek and palate, and although he can recognise touch over the left fauces and tonsil he states that it is much more numb than the right side. Palatal movements are perfect.

Comment.

The glosso-pharyngeal nerve supplies the middle ear, mastoid air-cells, and Eustachian tube by means of the tympanic nerve. This leaves the petrous ganglion actually inside the jugular foramen, and does not appear outside the skull. It follows that this branch is inaccessible, and pain arising in it can only be stopped by an avulsion at the base of the skull, which will probably tear the tympanic nerve as well; or by an intracranial section of the glosso-pharyngeal trunk before the branch has been given off. The pharyngeal supply of the nerve to the tonsil, posterior pillar of the fauces, and base of the tongue can be easily secured in the neck. Hence the cervical approach to, and extracranial division of, the nerve is more fitted for those cases in which faucial pain is dominant, but, even then, avulsion rather than clean section should always be performed.

My own case, on grounds of pure logic, was better fitted for the high (intracranial) section, but I judged his age to be against it, and fortunately the operation was entirely successful.

Purdon Martin's case showed the same limitation of pain to the tympanic branch in its initial stages, but for a shorter period, before extension to the throat; this was also the case in two of Doyle's patients. Martin compared this divisional electivity to that so common in trigeminal neuralgia, and my case enables me to confirm this view absolutely.

The innervation of the ear is complicated, and there might be doubt as to whether the glosso-pharyngeal through nervus tympanicus or vagus through ramus auricularis is the real cause. Sicard and Robineau expressed that doubt by the treatment they adopted for their cases (section of the glosso-pharyngeal, pharyngeal branches of the vagus, and excision of the superior sympathetic ganglion). But the syndrome of pain in the ear and in the throat can now clearly be regarded as glosso-pharyngeal, for clean intracranial division of the nerve has cured the pain absolutely. Crises of pure vagal disturbance have not been recognised save as a parasyphilitic phenomenon. The only disturbing feature about the crispness of the picture of aural and pharyngeal pain is Temple Fay's experiences with hopeless malignant disease of the pharynx, for in one of his cases pain in the throat was not relieved by a glosso-pharyngeal section, and in another was cured by cutting the vagus, the ninth nerve remaining intact. These were in examples of hopeless malignant disease, and run counter to the general mass of experience, such as it is. In my own view it seems to be certain that the vagus is chiefly motor to the pharynx and the glosso-pharyngeal chiefly sensory, no new idea but a reaffirmation of anatomical teaching borne out by clinical experience.

One other point is worthy of comment. It has been the custom amongst surgeons to explain the pain in the ear experienced by some sufferers from carcinoma of the tongue as referred along the lingual branch of trigeminus. But anterior lingual growths rarely produce it, and it is my impression that pain deep in the ear is only felt when the growth is far back and invades the fauces. It is therefore more likely to be glosso-pharyngeal than trigeminal. Pain referred to the tragus might be trigeminal, but not pain deep in the ear. After trigeminal sections there is surprisingly little anæsthesia in the auricle, and after glosso-pharyngeal section none at all, for it supplies the deep structures only. None the less, two or three cases of glosso-pharyngeal neuralgia in the literature have had painful ears, notably one case of Harris's. Nor is there any palsy of the soft palate or pharyngeal constructors unless the vagus has been coincidentally damaged.

Treatment.

As Harris has observed, and Martin supported the view, medicinal treatment may be successful for a long time. My patient obtained relief for years by drugs. No doubt these patients may have received drug treatment longer than trigeminal neuralgics, since the individuality of the affection has only recently crystallised out. But now that its identity is established recourse will be likely to be made to surgery rather earlier. As to the choice of route, there can be no doubt that the intracranial operation is the easier, and is therefore preferable. Even in the hands of those skilled in operations on the neck, the isolation and avulsion of the glosso-pharyngeal is a difficult undertaking and calls for gentleness and skill. In a thick-necked patient the nerve is very high and pharyngeal branches of the vagus may be mistaken for the nerve sought (vide Adson, case 2). Further the difficulty of ensuring section of the tympanic nerve is a disadvantage. It has succeeded, none the less, in Adson's hands and in my own. Temporary impairment of vagal functions may occur whichever route is employed, but fortunately does not seem to invalidate the result. Branches pass between the ninth and tenth nerves in the jugular foramen and avulsion of the former may cause slight damage to the vagus. The intracranial operation is essentially one for the surgeon familiar with the anatomy and methods of access to the lateral recess.

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BLOOD TRANSFUSION IN TWO CASES OF AUTO-AGGLUTINATION.

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THE phenomenon of auto-agglutination is not common, and transfusion in such cases is still more rare. We have not been able to trace, in the literature at our disposal, one case in which a transfusion was performed. In view of this it was thought advisable to place on record two cases which have recently come under our investigation.

In this department, although a grouped voluntary transfusion list is maintained, blood-matching is performed in every case before a transfusion is given. The serum of the recipient is matched against a red-cell saline emulsion of the proposed donor.[1] In both the following cases we found that the serum of the patient agglutinated the red cells of the donors. Some of the latter belonged to Group O (IV. Moss); there was therefore something more than the usual iso-agglutination in these cases. The blood-matchings were performed at laboratory temperature. At the suggestion of Dr. Dyke, the slides on which the matchings had been done were transferred to the incubator (temperature 37° C.), and it was found that the agglutination disappeared.

THE TWO CASES.

CASE 1, a married woman, aged 32, and seven months pregnant, had not been feeling well since the beginning of pregnancy. She complained of lassitude, headache, and noises in the head. Her appetite was poor, and there had been some recent swelling of the feet. There had also been some fairly severe hæmorrhage from piles. She was extremely pale and thin. The conjunctivæ were yellowish, and there was some slight œdema of the lower limbs. The heart was not enlarged but there was a soft apical bruit. The reflexes were somewhat diminished, but the plantar responses were both flexor. The interest centred around her blood condition. On admission on Jan. 24th, 1931, her blood count was:—

Red cells	1,040,000	Polymorphs	86 per cent.
White cells	8,000	Lymphocytes	13 ,,
Hæmoglobin	18 per cent.	Monocytes	1 ,,

Mean diameter of red cells approx. 8 μ. Six nucleated red cells in counting 100 white cells. Slight inequality in size of red cells.

With such a blood picture a transfusion was suggested, but it was found that the serum of the patient produced universal agglutination. The patient was herself Group B (III. Moss), and she had been tested against three donors of Group O (IV. Moss). When the slides were transferred to the incubator the clumping in all cases disappeared.

On testing the red cells of the patient against her own serum there was slight, but distinct agglutination at laboratory temperature, and intense agglutination at ice-chest temperature (4° C.). Agglutination also occurred between the patient's serum and the red cells of all other Group O (IV. Moss) individuals at 4° C., although this was slow in developing, but there was no agglutination when the matching was performed at incubator temperature. In view of the fact that the patient could not possibly agglutinate her own red cells at body temperature, a transfusion was agreed upon, her husband, Group O (IV. Moss), being used as the donor. Sixteen ounces of blood were given by the sodium citrate method, extra precautions being taken to maintain the blood at body temperature during the procedure.

The patient took the transfusion exceedingly well; there was no distress, and there was no appreciable increase in the pulse-rate. About twenty minutes later there was a sudden collapse, and the temperature rose to 104 F. She was extremely dyspnœic, but was able to tell us that she was suffering from pain and distress across her chest. Her condition gave rise to much anxiety. Tepid sponging was ordered, and morphia and adrenalin were given subcutaneously. These were followed almost immediately by cessation of all the symptoms, and there was no further period of anxiety. On the days following the transfusion there was no evidence of intravascular hæmolysis or clumping of the red cells, and no hæmoglobinuria or jaundice. The patient made a good recovery on preparations of iron. The following blood counts show the course of the convalescence:—

Jan. 27th: Red cells, 1,260,000 per c.mm.; reticulocytes, 10,800 per c.mm.
Jan. 31st: Hæmoglobin, 22 per cent.; red cells, 1,610,000; reticulocytes, 151,000.
Feb. 7th: Hb, 37 per cent.; red cells, 1,890,000; reticulocytes, 71,820.
Feb. 12th: Hb, 54 per cent.; red cells, 2,420,000; white cells, 9200; reticulocytes, 16,900.
Feb. 20th: Hb, 61 per cent.; red cells, 3,120,000; white cells, 10,300; reticulocytes, 15,600.

She was delivered of a full-time living child on

March 21st. A few days after her confinement she still showed the cold auto-agglutination of her own red cells, and also the red cells of Group O (IV. Moss) individuals.

CASE 2, a girl aged 5½, was one of thrombocytopenic purpura. A petechial hæmorrhagic rash was noticed five days before admission. It had started on the face and spread downwards. Two days before admission bleeding commenced from the gums and nose, and there was also some melæna and slight hæmatemesis.

On admission the child was very anæmic, blood was oozing from the gums and nose, and there was a generalised hæmorrhagic rash. The breath smelt of acetone. In order to arrest the hæmorrhage a transfusion was suggested. On doing the routine blood-matching we found that the serum from this patient agglutinated the red cells of each donor tested. The patient herself was Group A (II. Moss). Her serum had been put up against the red cells of her father, Group A (II. Moss), her mother, Group A (II. Moss), and those of another, Group O (IV. Moss). Agglutination occurred in all cases at laboratory temperature. The matching was repeated at incubator temperature, and the clumping disappeared.

The condition of the patient was desperate, and it appeared very doubtful whether she could stand a reaction such as was experienced by the patient in Case 1. In view, however, of the fact that unless the hæmorrhage were stopped death appeared inevitable in any case, a transfusion of 8 oz. of blood was given. The donor was the Group O (IV. Moss). The transfusion was taken well. About 45 minutes afterwards the patient collapsed. The temperature rose to 105° F., there was marked dyspnœa, and the child appeared to be suffering from severe shock. After our experience with the previous case adrenalin and morphia were given immediately, and tepid sponging was done. These were followed within a short time by complete recovery and cessation of the hæmorrhage.

On the following day the child developed a bronchopneumonia, probably aspiration in type. This ran a severe course, but the chest cleared completely in 12 days, and the child was discharged fit and well some ten days afterwards.

On admission the blood picture was as follows:—

Hæmoglobin	32 per cent.	Polymorphs.	71 per cent.
Red cells	1,520,000 per c.mm.	Lymphocytes	22 ,,
White cells	6,700 ,,	Monocytes	7 ,,

No morphological abnormalities of red or white cells; platelets, 1 per 1000 red cells; total, 1520 per c.mm. A few giant platelets noticed.

The prick which had been made in the lobe of the ear for this blood count was still oozing some three hours afterwards, but all bleeding ceased at the commencement of the rigor after transfusion. Subsequent blood counts were:—

May 14th (two days after the transfusion): Hb, 24 per cent.; red cells, 1,510,000; white cells, 13,200; platelets, 15 per 1000 red cells; total, 22,650 per c.mm.

May 26th (during the pneumonia): Hb, 29 per cent.; red cells, 2,010,000; white cells, 16,200; platelets, 146 per 1000 red cells; total, 293,460 per c.mm.

June 12th (two days before discharge from hospital): Hb, 54 per cent.; red cells, 3,730,000; white cells, 10,400; mean diameter of red cells, 7 μ; platelets, 88 per 1000 red cells; total, 328,240 per c.mm.

The patient was discharged in a perfectly healthy condition. The course of the convalescence was so satisfactory that there was no necessity to consider splenectomy, which Piney calls "the one almost certain method of inducing the onset of a thrombocyte crisis."[2] The giant platelets were noticed at the first blood examination only.

In this case some relatives of the child were also tested for auto-agglutination. The father and another daughter, both of whom were Group A (II. Moss), showed no auto-agglutination in the cold, but the mother, Group A (II. Moss), showed marked auto-agglutination at ice-chest temperature; this required fully two hours to develop. When the serum of the mother was put up against the red cells of Group O (IV. Moss) agglutination occurred rapidly at laboratory and ice-chest temperatures.

COMMENT.

The object of this paper is to show that cases which manifest auto-agglutination can be transfused. The recognition of these cases is rare; possibly they are much more common than is generally believed.

At a meeting of the Pathological Section of the Royal Academy of Medicine in Ireland,[3] W. Boxwell reported a case, and J. W. Bigger reported that he had been able to trace 22 cases of true auto-agglutination. Li Chen-Pien,[4] in an excellent article, describes his observations in such a case, and also gives a review of the literature.

The agglutination is not the usual iso-agglutination, but is apparently a temperature phenomenon and does not occur at body temperature. As a rule, the lower the temperature the more intense is the clumping of red cells. In view of this fact it is essential that the blood of the donor be not allowed to cool during the transfusion. In such cases the transfusion is apparently followed, after a short interval, by a severe reaction, but the symptoms can be relieved by morphia and adrenalin.

The experimental side of these cases has not been considered, as, at the present, this part of the work is incomplete.

SUMMARY.

1. Two cases of cold auto-agglutination are here described, one of anæmia complicating pregnancy and the other a case of thrombocytopænic purpura (Werlhof's disease). 2. In both cases transfusion was essential and was performed. 3. The transfusion in each case was followed by a period of extreme distress for the patient. 4. The symptoms of this distress were relieved by morphia and adrenalin. 5. In neither case was there any evidence of intravascular lysis of the red cells. 6. A thrombocyte crisis can be induced in cases of thrombocytopænic purpura by blood transfusion. 7. Auto-agglutination may be hereditary, as it was also present in both mother and daughter, Case 2, but it is impossible to formulate general rules on one case alone. 8. This phenomenon is not a manifestation incident to anæmia, as it was present after the anæmia had disappeared. 9. Group O (IV. Moss) still remains a universal donor. 10. Blood-matchings and blood-groupings should be done at body temperature and not at laboratory temperature as hitherto.

The blood-groups are given in terms of the international classification, those of Moss in parenthesis.

We are much indebted to Dr. S. C. Dyke, director of the pathological department of the Royal Hospital, Wolverhampton, who recognised the condition in Case 1, and to Dr. J. H. Sheldon and Dr. J. N. McTurk, visiting physicians, the Royal Hospital, Wolverhampton, for permission to use the clinical notes of these cases.

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OBSERVATIONS ON A SPHERULE RECENTLY DESCRIBED IN DISSEMINATED SCLEROSIS.*

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AND

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(From the John Burford Carlill and Neurological Research Laboratories, Westminster Hospital.)

THE spherule described by Miss K. Chevassut [1] as the specific virus in disseminated sclerosis, and its employment as the basis of a vaccine in the treatment of this disease by Purves-Stewart,[2] has given rise to considerable discussion during the past two years.

Smith and Ransome [3] also observed this spherule in cases of disseminated sclerosis, but did not have the opportunity of investigating other types of disease.

Carmichael [4] was unable to confirm Chevassut's findings, and a series of cerebro-spinal fluids submitted to her by him under test conditions were reported on in such a way as to cause him misgivings as to the validity of her original observations. Lépine and Mollaret,[5] of Paris, have recently published observations which still further throw grave doubts as to the spherule described by Chevassut being specific for disseminated sclerosis, since they have observed them in many other conditions. More recently still Tronconi,[6] of Pavia, confirms the findings of Lépine and Mollaret, and in addition to observing these spherules in liquid preparations has succeeded in obtaining them on agar slip-cultures.

Shortly after the original article by Miss Chevassut appeared we published the results of experiments on the biochemical and pathological effects of cultures of this spherule which were supplied to us by her.[7] In view of the experiments now to be described many of these deductions must be regarded as of little value.

As the result of a series of experiments in the nature of a repetition of the work of Chevassut, and before the publication of the work of Carmichael and Lépine, we began to have serious doubt about the specific nature of this spherule in disseminated sclerosis. This fact was made public with a promise of a fuller communication as soon as possible.[8] The present paper seeks to fulfil the promise. The preliminary experiments followed the lines laid down by Chevassut in her original paper, but the subsequent experiments were altered in the manner shortly to be described.

EXPERIMENTS.

Miss Chevassut in her first paper, and in subsequent communications, has stressed the fact that it is essential that the spinal fluid be received *direct* into the culture medium, or at the latest a very short time after. She claims that the discrepancy between her results and the diagnosis in the test series sent to her by Carmichael was due to delay in transmission of these fluids. Fluids from disseminated sclerosis were therefore investigated as follows.

One portion of each fluid was treated exactly as in the original technique, another was allowed to stand on the laboratory bench in the light for 48 hours, while a further portion was placed in the ice-box for a similar period, before being finally added to the broth and serum. It was found that whether the fluid was added direct to broth and serum, or whether allowed to stand on the bench or in the ice-box before being added to broth and serum, that on incubation these spherules appeared, the appearances being identical as regards morphology and number. It therefore does not appear that this objection of Miss Chevassut's is a valid one in the experiments carried out by Carmichael.

Stress has been laid on the necessity of using human serum, but rabbit's serum has been found to give quite as good results. Horse serum is not quite as efficient. The reaction of the broth has been stated to be of the greatest importance, and broth prepared by Hartley's formula was used in the original experiments. Ordinary tryptic broth, and even Lemco broth, gave almost equally good results, and one batch of quite roughly standardised Lemco broth seemed equally efficacious. Thus from these experiments many of the essentials laid down in the original paper seemed open to doubt. All attempts to obtain colonies on agar slip-cultures, after the technique of Welch [9] were unsuccessful both in cases of disseminated sclerosis and in all other fluids investigated, but Tronconi has succeeded in obtaining these in disseminated sclerosis and in other lesions of the nervous system.[6]

In the course of the preliminary investigations a considerable number of spinal fluids from lesions other than disseminated sclerosis were investigated, and spherules indistinguishable from those seen in disseminated sclerosis were observed.

The question of serum contaminants was not neglected, and it is admitted that these may present some difficulty; after a certain amount of experience these bodies can be differentiated from the spherules which form the subject of this paper. As a further safeguard (in the series of fluids to be subsequently described), a control tube of the same batch of serum and broth was incubated along with the serum-broth plus spinal fluid. In this way by examining drops taken from the control tube and the test mixture any appearances due to the spherule are readily appreciated. Further, it became a matter of constant observation that the controls remained quite clear to the naked eye while the positive tubes (after incubation for 24 hours to five days) presented a definitely turbid appearance. This turbidity, and therefore the appearance of the spherule, is caused, in our opinion, by the interaction of substances in the cerebro-spinal fluid and the blood-serum. Serum and broth alone do not produce this turbidity. Nor does it appear to be obtained from such cases of normal or hysterical patients that we have had the opportunity of examining. As it has not been possible to obtain a reasonable number of strictly normal fluids on which to make investigations, it would be wrong to express any definite opinion as to their constant appearance or absence in normal conditions.

It can be asserted that spherules, having apparently all the characteristics of the spherule described by Chevassut, can be observed in a large number of other abnormal conditions of the spinal fluid, and it is not possible to confirm her statement that they are specific to disseminated sclerosis. An examination of the Table shows that a positive culture can be obtained in all cases of organic nervous disease, when there is active breaking down of nervous tissue. This suggested that the spherules, granules, &c.,

* The major portion of this paper was communicated to the Pathological Society of Great Britain and Ireland on July 3rd.

are formed as the result of breaking down of blood protein by products of cellular disintegration—i.e., enzymes.

To test this possibility, a normal, fresh brain was stripped of its meninges, the grey and white matter separated as far as possible from blood-vessels, &c., and ground up with sharp sand. The resultant mixture was filtered through fine Berkefeld filters until it was optically clear. This extract was then used in a broth and serum mixture in the place of the cerebro-spinal fluid. In 24 to 48 hours an optical appearance consisting of spherules, granules, and filaments indistinguishable from that obtained in disseminated sclerosis was seen.

TABLE OF RESULTS.

	Cases.		
Cerebral thrombosis	1	....	Spherules present.
,, tumour	3	....	Spherules present in all.
,, abscess	1	....	Spherules present.
Spinal curvature	1	....	No spherules observed after 5 days.
Disseminated sclerosis	5	....	Spherules present in all.
G.P.I.	4	....	
Tabes	1	....	
Spastic paraplegia	1	....	Spherules present.
Parkinson's Disease	1	....	
Subacute combined degeneration of cord	1	....	
Amyotrophic lateral sclerosis	1	....	
Paralysis agitans	1	....	
Epilepsy	1	....	
Amentia	1	....	
Sciatica	1	....	
Uræmic fits	1	....	
Tuberculous meningitis	2	....	Spherules present in one case and absent in the other.

The Table is a list of 27 cerebro-spinal fluids treated under the same conditions. The conditions were that each fluid was exposed on the bench for 48 hours before being transferred to the serum-broth mixture (Hartley's broth, pH 7·6). This mixture was then passed through a small Berkefeld filter and incubated. A control broth-serum tube was similarly passed through a filter and incubated. Examinations of control and test cultures were made at intervals of 24 hours up to five days.

Thus, out of 27 cases, in which disseminated sclerosis only accounted for five of the series, 20 conditions other than disseminated were positive for the spherule and two negative.

CONCLUSIONS.

(1) The spherule described by Chevassut is not specific to spinal fluids of disseminated sclerosis, but can be found in the fluids from many other conditions. This agrees with the published findings of Lépine, Mollaret, and Tronconi. (2) Many of the essential conditions, especially that of the need for immediate transference of the spinal fluid to the broth-serum mixture, laid down in the original paper appear to be open to considerable elasticity on further investigation. (3) We have not been able to obtain agar slip-cultures, but this has been accomplished by Tronconi in disseminated sclerosis and in other lesions of the nervous system. (4) We do not consider the spherule to be a virus, but a flocculation or precipitation phenomenon produced by the interaction of the spinal fluid and blood-serum. (5) It has not been possible to study a sufficient number of normal fluids in order to state whether this phenomenon indicates an abnormal fluid or whether it occurs with any fluid.

In conclusion, it is desired to thank the physicians of the Westminster Hospital and of the Springfield Mental Hospital, Tooting, for their assistance in supplying fluids for these investigations.

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CLINICAL AND LABORATORY NOTES

DIPHTHERIA TOXIN OF HIGH VALUE.

By C. G. POPE, B.Sc. BRISTOL,

BIOCHEMIST, WELLCOME PHYSIOLOGICAL RESEARCH LABORATORIES.

FOR a number of years we have steadily increased the strength of the diphtheria toxin produced at the Wellcome Physiological Research Laboratories, and I feel it would be of interest to place on record the details of one batch of toxin of unusually high value. Working with tryptic digest medium, we have frequently obtained toxins with an Lf. value of 40 units per c.cm.* Recently a medium prepared from horse-muscle infusion and proteose peptone, containing sodium acetate, and sterilised by filtration through a Berkefeld candle—both important points—gave toxin with an Lf. value of 50 units per c.cm. This toxin was examined at the National Institute for Medical Research by Mr. Percival Hartley, D.Sc., who obtained the following results: M.L.D., 0·00038 c.cm.; Lo., 0·022 c.cm.; L+, 0·026 c.cm. I hope shortly to publish full details of the method of preparation of this medium and of others which have been found to give high-value toxins.

I wish to express thanks to Dr. Hartley for the tests which he carried out on this toxin, and to Dr. R. G. White for information regarding the methods used for preparing diphtheria toxin at the Belmont Laboratories.

* The abbreviation Lf. is a descendant of Ehrlich's Lo = Limes null—i.e., the dose of diphtheria antitoxin which, when mixed with one test dose of toxin, and injected into a guinea-pig, will prevent the appearance of any local œdema; similarly L+ is the amount of antitoxin which just fails to prevent the death of an animal. Lf. is the amount of toxin which (f) flocculates with one unit of antitoxin when mixed in the Ramon method. —ED. L.

ANTITOXIN OF EXCEPTIONAL POTENCY.

By A. T. GLENNY, B.Sc. LOND.,

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IT is of interest to other workers to place on record instances of antitoxin of exceptional potency. Among a group of 52 horses recently immunised with diphtheria toxin containing an average of 25 Lf. units per c.cm., prepared by Mr. C. G. Pope, seven produced serum containing from 2000 to 2500 units of antitoxin per c.cm., three between 2500 and 3000, and one contained 3000 units per c.cm. As this is the strongest unconcentrated antitoxin yet produced at the

Wellcome Physiological Research Laboratories, we sought an independent confirmation of our test; Mr. Percival Hartley, D.Sc., kindly offered to test this serum in the Department of Biological Standards, National Institute for Medical Research, and confirmed our findings. We have also to acknowledge the kindness of Mr. P. Bruce White, of the same department, in comparing the value of two samples of *Bacillus welchii* antitoxin, one an unconcentrated serum containing 800 to 900 units per c.cm. and a concentrated sample (produced from lower value sera) with 1300 units per c.cm.

The advantage of strong toxin cannot be too greatly emphasised, not only for the production of high-value antitoxin, but also for use as test toxin. The accuracy of any test can almost invariably be increased by the use of larger test doses of toxin. The use of a test dose of diphtheria toxin equivalent to 10 or 50 units of antitoxin increases the accuracy of titration, and tetanus antitoxin can be tested with advantage at the 2 rather than at the 0·2 international unit level.

INTRAMUSCULAR INJECTIONS OF CALCIUM.

By T. Izod Bennett, F.R.C.P. Lond.,
physician to out-patients, middlesex hospital.

The administration of calcium for the treatment of tetany has always been complicated by the tendency of calcium salts to produce necrosis on injection. This can be overcome when the intravenous route is employed, but makes the use of intramuscular injections for this purpose very difficult, if not impossible. Although in cases of acute tetany the intravenous method is the one of choice, it is not readily applicable in chronic cases.

I have recently been confronted with a severe case of chronic tetany, due to persistent steatorrhœa, which could not be controlled by the oral administration of calcium owing to defective alimentary absorption. It was, however, successfully controlled by the intramuscular injection of tetanol, a solution of calcium gluconate and lævulinate prepared by the Crookes Laboratories.

For the special purpose of this particular case, Messrs. Crookes prepared ampoules containing 5 c.cm. of a 3 per cent. solution of this substance, and these were given daily into the buttocks for a period of three weeks. These 21 injections were not attended by pain, and upon only one occasion was any local inflammatory reaction noted; the substance was rapidly absorbed, and arrest of the tetany successfully secured.

Enlargement of Isle of Wight Hospital.—Princess Beatrice recently opened extensions of this hospital at Ryde which have cost £16,000. They include a new women's surgical ward of 12 beds, an enlargement of the X ray department, and improved accommodation for nurses and the domestic staff.

Preston Infirmary.—Last year the in-patients at this institution treated to a conclusion numbered 5367, an increase of 617; there were 23,507 new out-patients, an increase of 2129. Casualties numbered 8573, and in the four theatres 6388 operations were performed. Owing to the industrial depression the Workpeople's Infirmary Fund produced nearly £2000 less, and 1930 closed with a deficit on the year of £5170. The average annual cost of a patient was £109 10s., the weekly cost £2 1s. 8¾*d*., and the cost per patient £5 4s. 7¾*d*.

BRITISH MEDICAL ASSOCIATION.

ANNUAL MEETING IN EASTBOURNE.

SECTION OF PATHOLOGY.

At the third session of this section on July 24th the chair was taken by Dr. R. G. Canti.

Epidemics.

Prof. Major Greenwood read a paper on the Factors Determining the Difference between an Epidemic of one Disease and that of Another. He pointed out that almost every word in his title alone required definition: what was a disease; what was an epidemic? Nevertheless, dialectical difficulties should not deter them from discussing a question which had interested the profession for thousands of years. According to Galen, there were three factors: the innate, temperamental and procatarctic, and the external atmospheric katastasis. In Galen's opinion it was rather the condition of those exposed to infection than the nature of the infection which determined the difference between epidemics. Sydenham, on the other hand, had enormously extended the sphere of the katastasis and popularised the doctrine of the epidemic constitution. In the year of grace 1931, epidemiologists were still wrangling about the problems of Galen and Sydenham. A via media must be chosen between the over-elaboration of the disciples of Sydenham and the over-simplification of academic bacteriologists. Experimental epidemiology was an attempt to simplify the study of general epidemiology by limiting the number of variable factors and by taking for study a herd of short-lived animals. These advantages were, of course, purchased at a heavy price, and the application of results to the human problems was analogical at two removes; there was a gap between the experimental mice and mice in general, and between mice and men. As a result it had been fairly well established that in herds of mice exposed to heavy risk of infection by *Pasteurella muriseptica* or *Bacterium aertrycke* the regular addition of healthy animals and careful exclusion of new infection would lead to the perpetuation of the disease through many generations. Though the infection might cease in small herds it would probably continue indefinitely in large herds. There was no conclusive evidence that environmental, non-specific factors modified the secular course of epidemicity in any significant way in these herds. Pre-immunisation greatly lowered mortality during the early herd life of the immunised animals, but ultimately the rates of mortality of pre-immunised and naturally immunised approximated to a level much above zero. Thus, it was unlikely that any artificially induced immunity would ever be more effective than that conferred in the ordinary course of herd life upon survivors of continuous infection. The very substantial advantage of pre-immunisation was that the survivors came into as good a position as the controls without having paid the price in high mortality, earlier on. No method so far tested would give a solid immunity—i.e., an approximation of the specific herd mortality to zero.

Prof. W. W. C. Topley agreed that the question set by the title could not be answered. The observation of "epidemic behaviour" over a large succession of waves might give a little information, but not much. It had been suggested—particularly by Boycott—that the study of mice in cages was not related to the facts of human disease: that mice did not bother to immunise themselves; that mankind depended much on immunisation, and that elephants

would probably be found to depend on it still more. This was unfair to mice, which were amazingly good at immunisation, both against mouse typhoid and against ectromelia. There was no solid immunity to the latter disease. Prof. Greenwood had made immunisation appear of some value against virus diseases, but he and his colleagues had not achieved the protection they had expected to achieve against bacterial diseases. Herd vaccination had lengthened the expectation of life but had not conferred much protection. At a guess, the diseases which were mainly toxæmic fell very nearly into the virus group as regards immunological behaviour. Data on the frequency of second attacks of diphtheria, scarlet fever, measles, and typhoid were remarkably scanty. On the other hand, the answer to the question of whether antitoxic immunity, however produced, was effective was probably in the affirmative. It seemed to be possible to secure good immunity against the streptococcus, but in scarlet fever the patients got tonsillitis; in puerperal fever it was possible to immunise against the toxin but not against the organism as such. The same was true of staphylococcal infections. Epidemiologists knew quite well the difference between diseases with different vectors, but it was nearly impossible to define the part played by each vector under clearly stated conditions. Prof. Topley confessed that he had absolutely no light on diet. A diet deficient in vitamins would doubtless increase the susceptibility, but the evidence was conflicting on whether a diet containing excess of vitamins would decrease it. Another unanswered question was whether this effect, if it occurred, was absolutely non-specific, or whether it favoured one sort of disease at the expense of another.

The variation in the virulence or epidemic potency of the parasite was probably a very important factor, but there was no valid evidence that it existed. Fluctuations according to virulence had been shown in the laboratory, but not under natural conditions. The existence of seasonal fluctuations was similarly doubtful. There was need of some means of measuring the relative importance of these different factors. Laboratory tests of immunity were very bad indeed, and far too much reliance was placed upon them.

Lieut.-Colonel C. A. GILL reminded the section that before 1919, when Prof. Topley had commenced his studies on mice, no progress had been made in epidemiology for 2500 years. Knowledge of the subject was at its very beginning. Moreover, epidemicity involved endemicity and ultimately the whole nature of disease. In his experience of malaria, plague, relapsing fever, and influenza, one of the most astonishing things was that, whether the disease was due to a bacterium and conveyed by an insect or whether it was conveyed by contamination in a laboratory, the epidemicity seemed to be entirely the same. Secondly, the old idea that an epidemic was due merely to some change in the specific parasite did not seem to hold water.

Dr. J. D. ROLLESTON stated that in his experience the proportion of recurrences of diphtheria under treatment remained constant at 1 per cent. He had seen only one second attack of small-pox, and second attacks were very rare in chicken-pox and typhus.

Dr. W. H. BRADLEY said that he had watched the throat flora in two waves of streptococcal tonsillitis in a school. Each wave had shown a uniform type, but the two types had differed, suggesting two definite waves produced by different serological strains. He exhibited graphs showing the tendency of a wave to stabilise itself after a while. Communities were, he suggested, saved from extermination in one epidemic by the onset of another, or, in nasopharyngeal disease, by the overgrowth of one strain by another.

The CHAIRMAN deplored the neglect of epidemiology, especially by the general practitioner. He asked what precautions should be taken on an outbreak of a disease with a high carrier and a low morbidity-rate, like meningococcal meningitis. It was impracticable to isolate all possible contacts.

Dr. W. G. BARNARD read a paper on

Infarction of the Myocardium.

This condition was, he pointed out, of general interest as the commonest cause of death in elderly persons. Knowledge of its morbid anatomy was necessary, because it might leave no visible signs in the heart and was frequently missed at autopsy. It could, however, be suspected partly from the history, and far more from the condition of the coronary arteries, which were blocked. Death was not a direct result of the block. By experiment on dogs it had been found that ligature of the coronary arteries caused fibrillation of the muscle, due to interference with the neuromuscular control through the cutting-off of the blood-supply. Necrosis and a degeneration of the myocardium could only be seen if the patient survived for a considerable time. If scissors or a probe were pushed down a coronary artery, it might easily be opened and a piece of atheromatous debris be removed without the operator finding the block, but if he made transverse sections he only required to know where the coronary arteries were. If the patient survived, the site of the lesion was marked by fibrous tissue, which might form a band or a series of lines and streaks which nearly always bore a deposition of clot. A certain amount of pericarditis was often found over the affected myocardium, which was nearly always in the left ventricle, where also aneurysms occasionally ruptured and caused hæmopericardium. The commonest cause of infarct in this region was atheroma of the coronary artery, with or without thrombosis. Emboli hardly ever occurred. In the rare younger cases an exactly similar condition might be due to aortitis. The orifices must always be examined carefully. Dr. Barnard showed a series of slides illustrating the post-mortem findings.

Dr. WILFRID MARSHALL read a paper on the

Spectra of Photo-electric Curves of Blood Pigments.

He said that the spectro-photometer gave accurate measurements of absorption in different parts of the spectrum and, if certain constants were maintained, would seem to be free from objections, although the curves did not represent the blood-spectrums seen by the eye. The instrument employed was a large-model constant-deviation wave-length spectrometer, with a polarisation photometer giving a triple field. Its average accuracy over the visible spectrum was about 1A. A convenient solution of blood with well separated oxy-hæmoglobin bands showed a clear blue and, on diluting the solution, the α band was the last to go. The curve of the absorption coefficients showed the β band denser than the α; the limit of the δ band towards the blue did not reach O, and was followed by increasing absorption as the violet was approached. This appearance was difficult to explain, but might be due to the residual yellow colour of blood solutions. Silver colloid solutions showed a similar tendency, and dilutions of electrargol were especially useful as a control. Dr. Marshall described experiments which seemed to show that a trace of methæmoglobin was normally present in the blood.

SECTION OF OBSTETRICS AND GYNÆCOLOGY.

Breech Presentation.

THE section met under the presidency of Mr. EARDLEY HOLLAND, and the first session was devoted to a discussion on this subject.

ANTENATAL TREATMENT.

Miss GERTRUDE DEARNLEY emphasised the need for prophylactic external version on account of the higher fœtal mortality in breech deliveries and the disadvantages to the mother which included exhaustion after long labour and severe lacerations of the pelvic floor. In addition, there was the need for ascertaining the relative sizes of the head and pelvis which was, of course, impossible with a breech presentation. The time of election was the thirty-fifth week, but if an anæsthetic was necessary it was better to delay version till the thirty-seventh week, in case premature labour supervened. An anæsthetic was unnecessary unless the patient was nervous or the abdominal muscles very strong. If the presenting part was engaged it was helpful to raise the foot of the bed for a short time before starting, or it might be necessary to push the presenting part out of the pelvis per vaginam. Most failures to turn were due to the neglect of this precaution. The speaker had never seen any serious complications follow external version, though in one case slight antepartum hæmorrhage occurred in a case of breech presentation with extended legs, where a good deal of force had to be used. The hæmorrhage soon cleared up and the patient went to term. It was best to turn the fœtus so as to maintain the attitude of flexion—namely, by pushing the head down towards the pelvis and away from the back—but, if this failed, movement in the other direction should be tried, and often succeeded. If the head did not sink into the pelvis, a binder should be applied. If version failed it was better to wait for a few days, and not infrequently spontaneous version took place in the interval. If the legs were extended, the important point was whether the feet were lying close to the head. If not, there was often quite good flexion of the body and version was usually possible under anæsthesia and without risk. If the legs were fully extended there was also extension of the child's body—points shown by X ray examination—and it was unwise to attempt version since damage might be done, and there was risk of bringing down a hand or arm by the side of the head.

FŒTAL MORTALITY AND INJURIES.

Mr. G. F. GIBBERD emphasised the high fœtal mortality in breech cases. In "uncomplicated" and "complicated" breech cases at a number of English hospitals whose last reports he had examined, the fœtal death-rates were 26 per cent. and 43 per cent., respectively. At Guy's Hospital the fœtal death-rate in primiparæ and multiparæ was 28 per cent. and 15 per cent., respectively, in uncomplicated breech deliveries. He did not classify first pregnancy or extension of the legs as complications. Amongst complicated breech cases, including in this class such conditions as prolapse of the cord and placenta prævia, the mortality was as high as 57 per cent. and 49 per cent., respectively. The most important cause of intranatal fœtal death was intracranial hæmorrhage, which accounted for 75 per cent. Next in importance was asphyxia, following cord compression after birth of the buttocks, and this accounted for almost all the remaining 25 per cent. He thought that the risk of drowning from premature respiration had been much overrated, and that a hot towel over the body during birth was useless. Injuries to the limbs were nearly always the result of bringing down extended arms which might cause either fracture of the humerus or an Erb's paralysis. The latter should be treated by putting the arm into the Israelite position, and allowing active movements daily when the splint was removed for bathing purposes. A fracture of the humerus was best treated by a straight Thomas's splint with elastic extension. Within a week or ten days firm union had taken place and the splint could be discarded.

MANAGEMENT OF BREECH LABOUR.

Mr. ALECK BOURNE said that at Queen Charlotte's Hospital the fœtal mortality in breech labour was 11 per cent. in primiparæ and 8 per cent. in multiparæ. One of the commonest causes of obstruction in breech cases was extension of the legs, and he believed that in every case in which this complication was present, if the child were of average or large size, one foot at least, and often two, depending on the size of the baby, should be brought down as a prophylactic measure as soon as the os was sufficiently dilated to admit a hand. Having brought down the legs, they should be folded as far as possible in the normal position by the buttocks, in order to get wider dilatation of the cervix. It was of great importance, however, to refrain from pulling at this stage. He thought that the most serious complication in breech cases was extension of the arms, because of the difficulty often experienced in flexing them and of the necessity for haste. Everything should be done to avoid extension of the arms, and the cases fell into two groups. In the first, the extension was "primary"—that is, the arms were extended when the legs were extended as a fœtal attitude before labour began. Over this group we had no control. "Secondary" extension was produced during labour entirely by pulling on the fœtus, and before the elbows could be reached and hooked out from the perineum. An incompletely dilated os predisposed to this, and it was for this reason that he advised folding the feet in the normal position by the buttocks. In that position they formed a larger mass than the breech alone, with both legs either extended or brought down, and, hence, caused better dilatation. In bringing down extended arms, he had found it useful to use a manœuvre of Potter's—viz., to press the lower angle of the scapula inwards. This brought the humerus downwards within reach of the fingers. In dealing with the aftercoming head the commonest mistake was to deliver it too quickly, especially if the head was not well flexed, the sudden moulding commonly leading to intracranial injury. The inspiratory movements of the fœtus at this stage were of little importance and were no indication for unduly hastening the birth of the head. The commonest cause of difficulty with the head was incomplete flexion, and this was especially liable to occur if the legs and arms had been extended, or if there were slight flattening of the pelvis. To obviate it, firm pressure should be made on the fundus during delivery of the body, combined with jaw traction with the index finger in the mouth, the body lying astride the forearm and the first and second fingers placed over the shoulders on either side of the neck. Before any traction was made on the shoulders the head should be flexed by gentle traction on the lower jaw and simultaneous firm pressure on the fundus. He had no experience of the use of forceps on the head.

Prof. F. J. BROWNE thought that version in breech cases was not always a safe operation for the fœtus,

There was a risk, if much force were used, of premature separation of the placenta, or the cord might become entangled around the limbs, while alarming slowing of the fœtal heart after version, probably due to shock, was a well-known phenomenon. He had seen two cases of face presentation follow version, and he thought this might easily be produced if the fœtus were turned so as to cause extension. He disagreed with Mr. Bourne as to the advisability of bringing down a foot as a prophylactic measure when the legs were extended, preferring to interfere only if and when need arose, because of the necessity for getting the fullest possible dilatation of the cervix and vagina in order to obviate, as far as possible, difficulty from extended arms or the after-coming head. In his opinion, these, and not extension of the legs, were by far the most serious difficulties in breech cases. He strongly advocated the application of forceps to the after-coming head if moderate jaw-and-shoulder traction failed to deliver.

Mr. DONALD ROY said that in the absence of large size of the child, a small pelvis, or uterine inertia, extension of the legs was not a frequentcause of still-birth; much more important was extension of the arms and head. To bring down the legs was no remedy for this, and might, in fact, by allowing the breech to slip quickly out with soft parts unstretched, increase the difficulty. If external version failed when the arms and legs were extended, and if there was anything to suggest from the appearance or history that the child was large, induction slightly before term was advantageous. In any case, where the child was large, the pelvis small, or inertia present, and especially if the legs were extended, one leg should be brought down early in the second stage, and right down as an excitant of good pains. Extension of the arms should be anticipated in such cases, and the arms in all cases felt for after the umbilicus was born. If not flexed, they should be brought down without delay, after which delivery of the head might have to be hastened by careful jaw-and-shoulder traction, assisted by efficient suprapubic pressure. Respectable results in breech delivery could now be obtained by labour in such cases taking place where people experienced in breech delivery were always available. This was difficult outside institutions or maternity homes staffed by efficient midwives. Mr. Roy quoted figures relating to 107 cases treated at the General Lying-in Hospital, York-road. There were 46 primigravidæ and 61 multiparæ, and the still-birth and neonatal death-rate was 6·5 per cent. in the former and 8·2 per cent. in the latter.

Dr. CHALMERS FAHMY said that at Edinburgh Royal Maternity Hospital the fœtal mortality was 22 per cent. and 21 per cent. in primiparæ and multiparæ, respectively. This mortality was very high, but it must be remembered that the cases were mainly delivered by somewhat inexperienced house surgeons. It would be interesting to know the fœtal mortality in 1000 cases delivered by practitioners of several years' experience. Extension of the legs was not in itself a serious cause of fœtal death. Delay in the arms and the head was the main cause. He did not think it wise to pull down extended legs as advocated by Mr. Bourne, believing it better to interfere only when necessary. In bringing down extended arms it was most important to wait till the axilla was almost in view; to attempt to release the arms earlier caused difficulty and increased the risk to the child. Fundal pressure should always be gentle. It could never flex an extended head; this could only be done by the finger in the mouth. Strong fundal pressure might cause intracranial hæmorrhage, and only jammed the extended head, as a whole, into the brim.

Dr. W. H. F. OXLEY quoted statistics to show that only about half of all breech presentations occurring in pregnancy were turned, and that attempts at version chiefly failed in the cases with extended legs. The difficult cases, therefore, still remained, and obstetricians were getting only half the experience in breech deliveries that they obtained before prophylactic version was introduced. He thought that better results, on the whole, would be obtained if we directed our attention to improving our skill in extraction, and left all cases unturned in order to provide more material for teaching and practice. He described in detail the method used in breech delivery at the East End Maternity Hospital. A team of three was necessary—midwife, obstetrician, and anæsthetist. When the os was fully dilated he ironed out the vagina and extracted the fœtus, very much in the manner advocated by Potter.

Mr. ERIC STACEY advocated the introduction of a speculum to hold back the posterior vaginal wall. The vagina was then swabbed out, and the fœtus thus allowed to breathe or even cry. One could then effect delivery at leisure. He thought Cæsarean section was justified in elderly primiparæ if the legs were extended.

Prof. J. B. DAWSON emphasised the need for external cephalic version in multiparæ, even if previous labour had been easy, on account of the tendency for the children's weight to increase in succeeding pregnancies.

Dr. J. C. MOIR thought that anæsthetics for version were dangerous, especially if too deep or given by an unskilled person, and that ether was more dangerous than chloroform. He disagreed with Mr. Bourne as to the reason why extended legs caused difficulty and delay in labour. It was not, as held by Mr. Bourne, owing to the greater cross section of the fœtus when the legs were extended, but because the lower limbs formed rigid bars which could not bend laterally, and so get round the curve of the pelvic canal.

The PRESIDENT said that the acid test of a good obstetrician was the way in which he conducted a breech delivery. Every hospital should have a senior resident medical officer, who could look after the delivery of difficult cases at any hour. All cases should be delivered on the back, and episiotomy should always be done if any difficulty were expected. If this were done no breech case should be lost, for it was the compression by the vulval ring that so frequently caused intracranial damage. He totally disagreed with Mr. Bourne on the question of bringing down a foot as a prophylactic measure. Bringing down an arm was a difficult procedure that involved six or seven different movements, in which all doctors should be thoroughly skilled. He believed that external version should be done in every case in which it was possible.

DISTRESS WARRANT AGAINST A HOSPITAL.—At Newcastle-on-Tyne Moot Hall police-court recently the Newburn rating authority applied for a distress warrant against the Newburn and District Cottage Hospital (14 beds) for arrears of rates amounting to £31 3*s*. 4*d*. Mr. H. Redhead, the secretary of the hospital, explained that when the Newburn Steel Works closed down last year the hospital lost 1500 weekly subscriptions of 1½*d*. each from workmen, and that the assessment had been increased from £8 10*s*. to £40. The house surgeon had been working for a long time without pay, and the hospital had kept going only by the exercise of drastic economy. The bench issued the warrant, which is, however, to be withheld, the hospital agreeing to pay £10 down and £2 per month.

REVIEWS AND NOTICES OF BOOKS

The Practical Treatment of Diabetes.

By T. IZOD BENNETT, M.D., F.R.C.P. Lond., Physician with charge of Out-patients, Middlesex Hospital, Physician to the Royal National Orthopædic Hospital, formerly Beit Memorial Research Fellow. London: Constable and Co., Ltd. 1931. Pp. 107. 6s.

Dr. Izod Bennett has written a small book which will be very useful both to the general practitioner, who is faced with the charge of a patient suffering from diabetes, and also to the patient. The doctor can use other and bigger books if he wants to, but the choice of a suitable book for the patient is a difficult one; the medical details necessary for the doctor are apt to frighten a patient.

The necessary information about the amount of sugar in the blood and urine is given in a simple manner, under the title of diagnosis; most of the rest of the book is devoted to the details of the treatment with and without insulin. Dr. Bennett thinks that the initial treatment should always be started with a fast day, and the diet rapidly increased up to the suitable amount. If the glycosuria persists, or the blood-sugar remains too high, insulin should be used. The rules for its administration are clearly set out, and the technique of the injection is faultless. The description of complications requiring operation, and of the effect of pregnancy, are very clear. The amount of food recommended for the average patient is 85 C., 110 P., and 110 F., and the question is discussed whether the diet should be changed to one containing 100 or more grammes of carbohydrate, with proportionate reduction in the amount of fat. Useful diet tables are given on the 5-gramme basis.

Recent Advances in the Study of the Psychoneuroses.

By MILLAIS CULPIN, M.D. Lond., F.R.C.S. Eng. Lecturer in Psychoneuroses at the London Hospital Medical College. London: J. and A. Churchill. 1931. Pp. 348. 12s. 6d.

THE "Recent Advances" series is well known and justifiably appreciated, but a volume on the psychoneuroses presents peculiar difficulties and cannot be brought completely into line. The author felt that the subject was so vague and beset with such prejudices that his book is more a review of the whole development of psychotherapy than a simple and straightforward account of recent contributions to an accepted department of medicine. He has, however, tackled his difficult task courageously, and with a considerable measure of success. After a brief historical outline, he discusses at some length the psychoneuroses of the war, from the experience of which his own interest in psychotherapy, like that of the great majority of workers in all countries, was derived. He then surveys the present position of psycho-analysis in a chapter which will be of interest to the novice, but which is not likely to satisfy the psycho-analyst, as it confines itself to elementary principles. Possibly the author regards the recent work of this school as too technical for the book as he planned it.

Succeeding chapters deal with various theories of memory, the conditioned reflex, and physical concomitants of emotion, and a good deal is said about asthma. The differential diagnosis of the anxiety, hysterical, and obsessional states is dealt with fully and clearly. Dr. Millais Culpin is exceptionally well at home in the chapters on occupational neuroses and the psychoneuroses of industry, and these two chapters constitute an excellent review of modern work in this important branch. But the chapter on the analytical psychology of Jung, written by Dr. James C. Young, seems out of place. It is an interesting expression of personal opinion, but it fails to set out the contribution which Jung makes to clinical psychotherapy and the important respects in which his technique and approach differ from those of the Freudian or Adlerian schools. It is in strong contrast to the accompanying chapter on individual psychology by Dr. A. R. Redfern, which sets forth very distinctly the point of view and method of Adler. The two remaining chapters are also by independent authors: Dr. Emanuel Miller discusses the psychopathology of childhood in a interesting and well-informed essay, with a valuable bibliography, and Dr. J. R. Rees gives a very useful guide to the establishment and maintenance of the psychotherapeutic clinic.

This volume, if it does not give a complete account of the recent progress of psychotherapy, at least records many important advances, and presents the general practitioner—for whom the series is especially designed—with a good introduction to this most troublesome subject.

Pratique médico-chirurgicale.

Third edition. Published under the direction of A. COUVELAIRE, A. LEMIERRE, and CH. LENORMANT. Paris: Masson et Cie. Tomes i. to iv. 2 vols., Fr.300; complete work, 8 vols., Fr.1200. Pp. 8500 approx.

WE have now received the first four volumes of the third and much-altered edition of this French handbook of the Practice of Medicine and Surgery. The first two editions were published under different editors in 1907 and 1911, and the present work is almost entirely a new undertaking. When completed it will consist of eight large volumes in attractive binding, containing articles collected for the special purpose from some 200 collaborators. The object of the publication has been to provide a complete account of the practice of medicine and surgery in all their branches for the use of practitioners and students, with particular regard to clinical methods and treatment, and to enable the doctor rapidly to obtain practical information on diagnosis and treatment, with little attention to theoretical or hypothetical problems. For this purpose the subject matter is arranged alphabetically under the various headings of diseases, forms of treatment, drugs, and the like. The whole work has been written and printed in the course of a year, so that articles are strictly contemporary. Many well-known names figure among the list of collaborators, and the whole is in fact a remarkable exposition of the present-day practical French teaching.

The arrangement of subjects in alphabetical order carries certain disadvantages, and an encyclopædia of medicine is by no means the most suitable type of text-book for the student. On the other hand, if the titles are well chosen this arrangement enables the practitioner to find the information he requires quickly and easily. In these volumes the subjects

have strange bedfellows. Successive articles in one group concern imbecility, immunity, impetigo, impotence, and in another anxiety, aortitis, aperitive, aphasia. Headings thus include diseases, symptoms, drugs, operative and other treatments, as well as more general matters such as auscultation, hygiene, epidemiology, and the like. Important subjects such as jaundice, diabetes, and arthritis are fully and clearly treated, and the notes under the names of drugs are excellent. Some of the numerous diagrams and pictures look rather old-fashioned. Some random attempts to test the completeness of the work failed to reveal any serious omissions. English readers will find much that is new to them in this work, which is a valuable book of reference to French medical practice.

Materia Medica.

Pharmacy, Pharmacology, and Therapeutics. Twentieth edition. By WILLIAM HALE-WHITE, K.B.E., M.D., LL.D., Consulting Physician to Guy's Hospital. Revised by A. H. DOUTHWAITE, M.D., F.R.C.P., Assistant Physician to Guy's Hospital. London: J. and A. Churchill. 1931. Pp. 712. 10*s.* 6*d.*

THERE must be few, if any, members of the medical profession in this country who have not heard of Hale-White's "Materia Medica." First published in 1892, it achieved a success so remarkable that from 1897 to 1905 a new edition was brought out each year. Since then fresh editions have appeared at frequent intervals, and the task of revision has now been handed over to Dr. Douthwaite. No alteration has been made in the characteristic features of the volume, which gives a full account of the preparations, action, and uses of all the official and many of the non-official drugs. It has been brought up to date by the introduction of drugs, such as ephedrine, avertin, and parathormone, which have come into favour during the last few years. A more detailed account of these might, with advantage, have been given, even if it entailed shortening the discussion of a number of others of less importance. There are a few curious errors in spelling—e.g., Novasurol is consistently spelled with two "r's," and Yohimbine with two "e's." We anticipate that the new edition will be as successful as those in the past.

International Clinics.

Volume IV. Fortieth Series. London: J. B. Lippincott Company. Pp. 309. 12*s.* 6*d.*

THE fourth volume of the Fortieth Series of International Clinics maintains a high level of interest. Three of the clinical papers come from France, one on the treatment of boils and carbuncles by injections of oxygen, another on the importance of the nervous system in the defence of the body against disease, and a third on rheumatism of dental origin. The staff of the pædiatric department of the Fifth Avenue Hospital of New York City present contributions on child health, on the treatment of weak feet in children, on procedures in the prevention and correction of oral deformities in children, and on a comparative study of posture in the first and second decade of life. The general staff of the same hospital have clinical contributions on the care of patients in the terminal stages of cancer, on toxin-antitoxin immunisation for diphtheria in adults, postpartum fever, and allergic purpura, while there are papers by other authors on hereditary epistaxis, on the differential diagnosis of peptic ulcer, and on subacute recurrent duodenitis. Among the most interesting features of this volume are reprints of some post-graduate lectures arranged by the Medical Society of New Jersey. Prof. J. E. Talley, of Philadelphia, deals with the treatment of cardiac diseases; Dr. T. Fitzhugh, Jr., of the same city, gives a review of modern advances in diseases of the blood; and Dr. Harry Gold, of Cornell, considers some of the larger problems of drug therapy. Dr. Gold points out that one of the stumbling blocks to the successful use of drugs is the question of dosage. The physician tends to think of the dosage of a drug in terms of weight or measure rather than in terms of activity or the effects it is likely to produce, with the result that often too small amounts are administered. As a general rule, in his view, no patient should be regarded as having been adequately treated with a drug unless the full therapeutic effects have been obtained, or minor toxic symptoms produced. There can be no question but that this is a sound general principle in therapeutics, though it requires some qualification, for there are many drugs which in toxic doses are definitely dangerous and, moreover, delayed effects may have to be reckoned with. At the same time the dangers can be reduced to a minimum if the physician has a working familiarity with the earliest signs and symptoms of toxic action and therapeutic effect. This is, in fact, though Dr. Gold does not state it explicitly, a strong argument for more attention being paid to the clinical aspect of pharmacology in the training of the student.

Wilhelm Conrad Röntgen

und die Geschichte der Röntgenstrahlen. By Dr. OTTO GLASSER, Cleveland Clinic Foundation. Berlin: Julius Springer. 1931. Pp. 337. R.M. 29.60.

Dr. Glasser has performed a task which is particularly opportune at this time, when we are removed from the glamour of the early days of radiology, and are yet, fortunately, still in touch with many who took part in, and can remember, its inauguration. The author has spared no pains in collecting information about the discoverer of X rays and his work, and he includes lighter touches in the form of contemporary cartoons and advertisements in the public press. It is a story full of romance. So many stopped on the threshold of discovery, notably Crookes, in 1879, and other workers who were using Crookes and Lenard tubes in the years immediately preceding 1895. All this is set out by Dr. Glasser fairly and fully.

It is remarkable to look back on this early history, and to see how large a part of the subject, as we know it, was covered within the first year of the discovery. There is, for instance, a reproduction in the book of cinematographic pictures of the frog's leg made by Dr. James MacIntyre, of Glasgow, and published in the *Archives of Skiagraphy* for 1897. Now almost forgotten is the metal X ray tube made in March, 1896, which lies in the collection of the British Institute of Radiology at the South Kensington Museum. In these days of elaborate apparatus, it is interesting to be reminded of such an advertisement as: "Set of superior X ray apparatus, as used at the Leeds General Infirmary, &c., complete sets, including 2½ inch coil, battery, and focus tubes, from £7 10*s.* 0*d.*"

The volume has interest for every worker, whether on the medical or the physical side of radiology. Although the author is resident in America, the book is written in German, and we hope that it will be made more accessible to English readers.

THE LANCET.

LONDON: SATURDAY, AUGUST 22, 1931.

THE NUTRITIONAL ANÆMIAS.

THE historian of internal medicine will probably consider that the most important advance of this decade was the clear recognition of the nutritional anæmias. Discoveries such as the isolation of insulin have led to the successful treatment of specific diseases, but the candle lit by G. R. MINOT and W. P. MURPHY'S work on liver in pernicious anæmia has thrown light on many different forms of obscure ill-health which arise from nutritional causes and have anæmia as an outstanding feature. In a paper on Simple Achlorhydric Anæmia at the front of this issue, Dr. D. T. DAVIES calls attention to the group of cases described by KNUD FABER, in Denmark, by MILLS, WATKINS, and others in the United States, and particularly by L. J. WITTS,[1] in Great Britain. To WITTS'S 50 cases Dr. DAVIES adds a similar group. The patient, usually a woman, suffers as a rule for years from indigestion and a progressive anæmia, characterised by marked reduction of hæmoglobin, but with little change in the red cell count. Achlorhydria is ordinarily present, but pepsin does not disappear from the gastric juice, and secretion of hydrochloric acid can often be demonstrated after injections of histamine. Treatment with large doses of the scale preparations of iron is usually very satisfactory.

The distinction between this type of anæmia and pernicious anæmia of the true Addisonian type is definite; the simple anæmia is almost confined to women, is insidious in its onset, taking many years to develop, is always marked by prolonged dyspepsia, is associated with a much reduced intake of food both as regards quantity and variety, produces a blood-picture essentially secondary in type, and responds quickly to administration of iron. Addisonian anæmia, on the other hand, is fairly rapid in its onset, affects both sexes, is often not associated with gastro-intestinal symptoms, is always accompanied by complete achylia gastrica, produces a blood-picture which is hæmolytic in type with macrocytes and marked reduction of the red cell count, and is not curable by the administration of iron. In spite of these contrasts there is, nevertheless, an aspect of close similarity between the two diseases in that both are associated with a reduction in gastric secretion and both may be considered as largely nutritional in origin. The work of W. B. CASTLE,[2] who showed that food subjected to the influence of normal gastric juice could produce striking amelioration in the symptoms of pernicious anæmia, has led to the introduction of extracts of gastric mucous membrane, whose beneficial effects have been almost as great as those of liver. It is believed that normal gastric juice can elaborate from protein of animal origin a substance which promotes normal blood regeneration; its exact composition is at present unknown, but it is neither hydrochloric acid, pepsin, nor any other recognised constituent of gastric juice. The school of thought which always laid stress on achlorhydria as the one constant finding in pernicious anæmia was formerly of opinion that the importance of the gastric change lay in the fact that it permitted bacteria to pass unchecked direct into the small intestine, but the more modern trend of reasoning is that achylia in pernicious anæmia connotes the absence of an essential but unknown chemical constituent of the gastric juice secretion. In simple achlorhydric anæmia, on the other hand, it is believed that prolonged dyspepsia with an ill-chosen diet, either as cause or effect, can, especially if hypochlorhydria be present, lead to such a defective assimilation of iron that the deficiency has direct and damaging results.

But the conception of nutritional anæmia extends far beyond these two diseases. The severe anæmia not infrequently seen after partial gastrectomy, or after the onset of chronic gastritis following gastro-enterostomy, has been described by G. GORDON-TAYLOR and others, and without providing a condemnation of gastric surgery, such observations serve as a warning against unnecessary reduction of gastric function. In this connexion it must not be forgotten that the pyloric mechanism is in itself a safeguard which tends to secure adequate nutrition. In all cases of achlorhydria without pyloric obstruction, the emptying time of the stomach is greatly accelerated, and the same effect tends to be produced when gastro-enterostomy or gastrectomy are performed: the result of this is not only that the food is under the influence of gastric juice for a much shorter time, but also that it reaches the small intestine too rapidly and in amounts too great for the digestive powers of that most important section of the alimentary canal. The effect is often apparent in the form of loss of fat in the fæces, and gas distention of the ileum and colon due to the hydrolysis of starch by microbic action instead of by the diastatic ferments of the succus entericus. Another form of nutritional anæmia is to be found in tropical sprue where, whatever the ultimate pathology may prove to be, the essential feature is failure of absorption by the alimentary canal. The result is that not only is fat prevented from reaching the blood stream in adequate quantities, but other essential substances, such as iron, calcium, and vitamins, are insufficiently absorbed, and a striking anæmia is one of the consequences. The Plummer-Vinson syndrome, in which dysphagia is added to the symptoms commonly seen in severe achlorhydric anæmia, is yet another instance of a disease in which malnutrition plays a major part, for it has been shown that even if the dysphagia is not the primary cause

[1] Guy's Hosp. Rep., 1930, lxxx., 253.
[2] Amer. Jour. Med. Sci., 1929, clxxviii., 748.

of the disease the symptoms undergo rapid amelioration when nutrition is restored to normal.

It is impossible to review the vast amount of work of which a brief sketch has here been given without seeing the significance of malnutrition in the genesis of disease which may be mild in character but is none the less of immense importance because of the large number of people affected. We have certainly made much progress since SAMUEL FENWICK'S discovery in the middle of the last century, that pernicious anæmia is associated with atrophy of the mucous lining of the stomach.

ALL ABOUT HOSPITALS.

THE Hospitals Year Book for 1931, which makes its appearance this week, is the first issue[1] of an annual record of the hospitals of Great Britain and Ireland, and within its pages there is incorporated the year book of philanthropy so familiar to us as Burdett's "Hospitals and Charities." This year book was issued for the first time in 1890, and for 40 years proved a valuable guide to the hospitals and asylums of the United Kingdom, to institutions for rest and convalescence, and to all the agencies which assist the poor and helpless. Throughout this long period of success, "Burdett" became larger and more detailed in its contents in response to the growth of the activities chronicled, but, in the meantime, the character of many of the voluntary hospitals, in whose behalf the publication was founded originally, became modified in two significant ways: first, contributory schemes of payment came gradually to play an increasing part in hospital finance, and, secondly, the unification of hospital and medical services under the Local Government Act brought about a fundamental change in the general picture. Accordingly, the publishers approached the Joint Red Cross Council and the British Hospitals Association, foreseeing that coöperation with these bodies should result in the taking over of "Burdett" as a substantial part of a complete survey of the hospital world in this country from year to year. The merging into a central bureau of the Incorporated Association of Hospital Officers, the British Hospitals Association, and the Joint Council of the Order of St. John of Jerusalem and the British Red Cross Society made a comprehensive policy possible. The new bureau now publishes, in this annual year book, all the information previously collected and issued by its constituent bodies, including the masses of general and particular information, comprised in Burdett's "Hospitals and Charities."

The beautifully printed volume, which has now appeared, will form the basis upon which to build the future issues, and, while the foundations are truly laid, the superstructure is symmetrical, requiring though the attention of the numerous authorities concerned in the provision of such an annual report, to fill up lacunæ. The importance of a compendious review of the work done by the voluntary hospitals and of the money spent on doing it, is recognised to so full an extent as to give a feeling of certainty that future editions will contain a complete picture of the institutional treatment of the sick in this country. Difficulties connected with the inclusion or omission of any particular institution should not arise, for no one would expect from the bureau the exercise of any discretion in the matter—every hospital should find a place in the compendium. The absence of an index is met by an alphabetical list of contents, which precedes an alphabetical arrangement of matter, and this is followed throughout, so that the editors will certainly hear of any omissions that may have crept in, despite their anxious attention. It is certain that the preparation of the large portion of the volume, forming a directory of the institutions, entailed great editorial labour, for alterations in the usual format of the publications of the constituent bodies of the bureau had to be made in conformity with the pattern in "Burdett," so familiar to all hospital workers, and it may be mentioned that many whose avocations would make necessary a frequent employment of the pages of "Burdett" have been consulted as to the particular directions in which the older publication has been found useful. But this arranging and rearranging of the hospital directory consumed a great deal of time, with the result that the statistics in the volume, which might have been published earlier, have undoubtedly somewhat lost their topical value. Such delay need not occur in future editions with a settled routine upon which to work. When we have said that the information connected with mental hospitals and municipal institutions will have not only to be amplified, but brought into line with that provided in connexion with voluntary hospitals, we exhaust our criticism of the volume in its present state. But the unity of objective and the fusion of interests which mark the two great classes of our hospitals will have to be recognised in the Hospitals Year Book.

Besides the hospital directory, the volume reprints many of the memoranda on matters of administrative interest to hospitals which have been issued from the bureau. A more recent memorandum, and a very timely one, on hospitals and motoring accidents is epitomised in the later pages of our present issue. The compilers responsible for an arduous piece of work, upon the fine outcome of which they deserve every congratulation, are as follows:—Mr. R. H. P. Orde and Mr. A. E. Ceadel, editor-in-chief and secretary, respectively, of the Central Bureau of Hospital Information; Dr. H. L. Eason, superintendent of Guy's Hospital, Mr. F. G. Hazell, superintendent and secretary of the Royal Infirmary, Manchester; Captain J. E. Stone, secretary of the Birmingham Hospitals Centre; Mr. Sydney Walton and Mr. Wynne Williams, representing the publishers; and Mrs. M. K. Cooper, editorial secretary for the large section of the work constituting a hospital directory. We regret to record the death of Mr. Wynne Williams on the day of the issue of the volume.

[1] London: Nursing Mirror, Ltd. 1931. Quarto. Pp. 639. 15s.

THE SPASTIC COLON.

UNDER modern urban conditions people pay increasing attention to the behaviour of their bowels, and practitioners hear more about constipation than any other trouble. The symptom has, of course, important organic causes, but these can account for 5 or 10 per cent. of the complaints at most. Many patients have no true constipation at all, but only dyschezia or rectal retention, while the majority have some purely functional disorder of the colon. In this last category a distinction is usually made between an atonic and a spastic constipation, and in many respects this is a good one. In practice mixed forms are commonly seen, and the spastic group especially includes a variety of clinical types. Thus Dr. E. I. SPRIGGS[1] divides them into colonic delay, irritable colon, spastic colon and colospasm, mucous colitis, and muco-membranous colitis. At least the four last types, however, have so much in common that from many aspects they may be considered under a single heading, as representing merely grades of the same disorder.

In the causation of the spastic state the nervous element is of the greatest importance. The normal tone of the colon is maintained largely by the nerve plexus in the wall of the bowel, and is controlled by the autonomic nervous system, the vagus being mainly augmentor and the sympathetic inhibitory. It is only necessary to recall the diarrhœa of fright or emotion to appreciate the powerful influence of the mind upon the motility of the bowel. In another way, too, it is possible that nervous factors may influence the colon—namely, through its blood-supply—the secretory abnormalities being thus accentuated. FLOREY and others [2] have shown that in the colon mucosa of dogs excitement causes blanching and pallor, from vaso-constriction, which may long outlast the initial stimulus. Thus, as Dr. SPRIGGS says, local alterations in circulation may well play a part in the pathology of these disorders. A further factor of essential importance is local irritation either within the bowel or elsewhere in the abdomen, including the appendix. Constipation as such may secondarily produce spasm of the colon, just as spasm of the colon of nervous origin may produce constipation. Moderate constipation alone, however, is probably far less dangerous to the colon than is its treatment by irritant purgatives. Among these Dr. SPRIGGS includes such familiar remedies as rhubarb, cascara, and senna, as well as saline aperients when used regularly. The frequency with which such drugs are taken is shown by an analysis he quotes in which some six out of every seven constipated subjects were found to be taking aperients, and more than half of them were doing so every day. Many cases of spastic colon may therefore arise through irritant treatment of a temporary harmless constipation, and the abuse of purgatives is undoubtedly a danger. Certainly inflammation of the mucous membrane by purging increases the liability to absorption of toxins or bacteria from the gut, and diarrhœa so produced does more harm to the general health than is done by simple constipation with its drier stools. Indeed bacteriological study of the fæces in constipation does not suggest that serious bacterial intoxication is common from this cause.

The symptoms of spastic colon are variable, especially since the spasm is often merely one manifestation of an underlying neurosis. Many of the patients are of nervous constitution, with a "highly strung" and irritable temperament. They commonly complain of undue fatigue, depression, and general indications of malaise such as we are apt to associate with intestinal intoxication—for instance, headache and giddiness. Some abdominal pain or discomfort is almost constant, and in severe cases pain may be paroxysmal and so intense as to simulate acute appendicitis or renal colic. Diagnosis must often be made finally by radiography, but it is well to beware of attaching too much importance to anatomical variations in the shape and position of the colon, and the standard of normal motility must not be too rigid. Treatment, once the condition is recognised, should be largely psychological. If the importance of the nervous factor is realised, and the constipation obsession can be removed, the battle is half won. Full reassurance should be given concerning the relatively harmless results of the bowels not being moved for two or even three days, and sources of worry and anxiety should be removed wherever possible. In general a change of environment at the onset of treatment is required, and later an occupation is desirable when, as sometimes, the patient has too little to do. Where, on the other hand, work and recreation are already too strenuous, they must be modified; for the spastic colon often arises from an over-excited, anxious, or "spastic" state of mind. Short periods of rest during the day may be helpful. The constipation itself must, if possible, be relieved without the use of chemical purgatives, and Dr. SPRIGGS thinks that nearly always the colon and the patient will recover if this can be achieved. Encouragement, regular habits, "re-education" of the bowels, and a suitable diet are the main points. Rectal douches should be used with discretion, but are often an important aid, as also is massage of the colon in certain cases. Of drugs the mineral oils, either by mouth or as small enemata to be retained overnight, with sometimes castor oil, and the use of belladonna are most usually advised. The so-called "roughage" diet is advocated by some physicians, but it seems more rational, as Dr. SPRIGGS recommends, to exclude foods such as wholemeal bread and vegetables which leave an undigested residue in the colon.

Its frequency, the diversity of its symptoms (mainly nervous), and its excellent prognosis with adequate recognition and treatment, make spasm of the colon very important, and Dr. SPRIGGS's review is one which deserves careful study.

[1] Quart. Jour. Med., July, 1931, p. 533.
[2] Drury, A. N., Florey, H., and Florey, M. E.: Jour. Physiol., 1929, lxviii., 173.

ANNOTATIONS

DRUGS AND THE PRACTITIONER.

In a course of lectures delivered to graduate students in New Jersey (and reviewed on p. 408) Dr. Harry Gold shows how easy it is, in the evaluation of therapeutic agents, to be led into serious blunders by inadequate experiments, insufficient controls, or unsuspected sources of error. A common mistake is to focus attention too closely on a single effect produced by a drug, without realising the numerous combinations of action which might give rise to it. The guanidine derivative, synthalin, for example, reduces the blood-sugar after oral administration, and was therefore employed and recommended in the treatment of diabetes. But a clearer understanding of its action showed that the effect is due to an injury of the parenchyma of the liver and not to an increased capacity of the body to burn sugar. The truth is that new drugs become widely used clinically before accurate knowledge of their action is available, and continue to be used blindly long after the truth is discovered. Dr. Gold utilises facts like these to drive home to the clinician in the U.S.A. the advantages of confining his therapeutic armamentarium in the main to the non-proprietary drugs listed in the book called "Useful Drugs," published by the Council on Pharmacy and Chemistry of the American Medical Association. For those wishing to give proprietary substances a clinical trial, he suggests they should confine themselves to drugs which are listed in the book entitled "New and Non-Official Remedies," published by the same Council. This work is well known in this country as well as in the U.S.A., but for those to whom it is unfamiliar Dr. Gold explains its scope. He says:

> "If a new drug is not described there or is found in the list of preparations considered but rejected, one can rest assured that the claims made for that preparation have not been substantiated by evidence that can be considered satisfactory to authorities of that subject. There are some exceptions in which preparations are rejected because they fail in other respects to comply with the rules of the Council of the American Medical Association. When a new preparation is submitted, the Council makes a careful analysis of the evidence presented to support the claims. The character of the investigation varies with the nature of the product and the claims that are made. In many cases chemical analyses are performed and pharmacological and clinical studies carried out by disinterested workers under the auspices of the Council for the purpose of checking the evidence. In most instances," says Dr. Gold, "it becomes necessary for the manufacturer to reduce his claims to conform with the results of these additional investigations, the findings of which are usually far less optimistic than the original claims."

The Council on Pharmacy, it seems, goes a little further than our newly appointed Therapeutic Trials Committee, which proposes to examine new products only on the application of commercial firms. No attempt however is made by the Council to dictate to the doctor what he is to use in his practice. The products accepted are not even recommended. All that is attempted is to aid the physician in doing what he is trying to do himself, but without adequate facilities—namely, to separate the great mass of unofficial medicines into two distinct groups, those with valid claims and those with claims which cannot be substantiated. There is, for example, no class of drugs in which it is more difficult for him to get at the truth than in the case of the barbituric acid hypnotics. It is about 30 years ago since veronal was introduced into practice, to be followed in the intervening years by a whole series of other derivatives of barbituric acid. The claims usually made for these hypnotics are that they are more effective therapeutically, and that they are safer than veronal. Yet, according to Dr. Gold, if these are tested by the "blind test," so as to exclude the influence of subjective factors, it appears fairly certain that the therapeutic and the toxic actions of the various members of this group run essentially parallel, and that whenever the chemical structure of barbituric acid has been changed so as to increase the hypnotic action, the intensity of the toxic action has been increased to practically the same degree. It follows therefore that if the various members of this group of drugs are given in doses comparable in terms of their relative potency, they have approximately the same safety and the same therapeutic efficiency. Nevertheless there remains more in the philosophy of therapeutics than is dreamt of in the most accurately compiled list of preparations whose potency is proven to the satisfaction of any school of pharmacology.

RECENT WORK ON EPILEPSY.

Epilepsy may or may not be a metabolic dyscrasia, as J. S. Collier has asserted, but its course can undoubtedly be influenced by metabolic factors. By starvation, by ketosis, or by dehydration epileptic fits may often be greatly reduced in number, and sometimes completely arrested. N. P. Walker and G. A. Wheeler[1] now claim a similar result from yet another metabolic manœuvre—namely, elimination of the pellagra-preventive factor from the diet. This was suggested by symptoms of pellagra in a patient on the ketogenic diet, the appearance of such symptoms being accompanied by a marked decrease in the number of fits. Ten chronic epileptics, all with considerable mental deterioration, were placed on a diet well balanced in all respects except that the pellagra-preventive factors were absent, or only present in very small quantities. After a varying period, averaging about four months, stomatitis and typical skin eruptions appeared. When the symptoms became severe, treatment by yeast was instituted, and readily caused their disappearance. Fit records show that about the time the pellagra symptoms appeared there was, in most of the cases, reduction of about 50 per cent. in the number of attacks. The reason for the reduction must at present be a matter for conjecture, but the observation may prove another useful piece of ammunition for those who are seeking to attack epilepsy on its metabolic side. For obvious reasons, the method is not suitable for ordinary practice.

The theory that epilepsy is often the result of accumulations of cerebro-spinal fluid, whose drainage is impeded by pathological processes in the Pacchionian bodies, we owe to Temple Fay. Much of his work is based on encephalographic studies. Other observers, however, have taken encephalograms of epileptics with less consistent results, and in the absence of a series of encephalograms of normal people, there is no substantial agreement as to what constitutes a normal picture. J. Notkin,[2] after studying 17 female patients who were suffering from idiopathic (cryptogenic) epilepsy, claims that a dilated ventricle and a large accumulation of air in the subarachnoid spaces must be regarded as pathological. In his method all the fluid that could readily be drained off

[1] U.S. Public Health Rep., 1931, xlvi., 851.
[2] Arch. Neur. and Psychiat., July, 1931.

by lumbar puncture was removed and replaced by air; the amount varied from 75 to 155 c.cm. After this an X ray appearance that he considered normal was obtained in eight cases; and in these most of the air had disappeared within 24 hours. In four cases there was pronounced dilatation of the ventricles and subarachnoid spaces, especially in the region of the Pacchionian bodies, and repeated pictures showed persistence of the air, in one case up to 216 hours later—a phenomenon which Notkin is inclined to associate with the ventricle dilatation. In the five remaining cases there was evidence of other pathological changes, such as asymmetry of the ventricles or leptomeningitic adhesions, pointing to inflammatory, or possibly neoplastic, processes, which were apparently not active. It is probable that further study along these lines would lead to results of high value; and this indeed is indicated in the consequent successful therapy claimed by Temple Fay. But a procedure such that no one has so far attempted to apply it to a series of healthy people should be approached with caution. Notkin is careful to inform us that none of the patients died as a consequence of the procedure; but one died five weeks later from a severe convulsion, and another eight weeks later from an unexplained hæmoptysis. All the patients had severe headaches for 48 hours after the operation, together with rigidity of the neck, profuse perspiration, vomiting, and tachycardia, which subsequently gave place to a pronounced slowing of the pulse. He is inclined to think that some of these symptoms might be obviated if the volume of air introduced were rather less than that of the fluid withdrawn.

INFANT DEATHS AND VITAMIN STARVATION

THE North Kensington Medical Society has circulated among its members an analysis of the causes of the 180 deaths of infants under one year which occurred in the borough during the year 1928. The report is the work of three practitioners, Dr. H. E. Barrett, Dr. A. Keith Gibson, and Dr. Eleanor M. Reece, and their conclusions are worthy of close scrutiny, as they differ appreciably from those usually reached in an inquiry of this kind. In each case investigated an attempt was made to classify the various environmental factors as "good," "fair," or "bad," these factors including housing, income, antenatal and postnatal maternal employment, feeding, maternal care, foster care, size of family, and the proximity of open spaces. In nearly 40 per cent. of the cases the family income was unsatisfactory, and an analysis of other factors in the group, taken as a whole, showed that environment deteriorated in direct proportion to the decrease of family income. In homes where the income was small, therefore, they assume that death was probably the result of several unsuitable environmental factors, or, in other words, that poverty was the real cause behind a number of these deaths. It was found, however, that bronchitis and broncho-pneumonia were no respecters either of houses or incomes. Among the children between six months and a year in age who died as a result of these respiratory diseases it was found that a large proportion had shown signs of rickets. The incidence of breast-feeding in this group, however, was high; out of 55 deaths from respiratory infections 30 infants were completely and 18 partially breast-fed. The investigators point out that those foods which contain vitamin D also contain vitamin A, and that a child who is showing signs of rickets must therefore be receiving a diet deficient in both these vitamins. Recent experiments on the resistance conferred against infection by vitamin A lead them to conclude that a deficiency of this vitamin would be sufficient to account for the occurrence of broncho-pneumonia associated with rickets in children between 6 and 12 months old. Even during the period from the second to the sixth month they consider a deficient diet may be responsible for deaths from respiratory infections, and claim that great improvement has been seen in cases of bronchitis and broncho-pneumonia when vitamin preparations have been administered along with the ordinary remedies. Deaths under one month from these conditions may be due, they suggest, to an innate tendency to infection, due to faulty diet and environment of the mother during pregnancy. Anticipating that the high proportion of breast-feeding in this group may be quoted as an argument against their theory, the investigators point out that it is now well established that the quality of breast milk and its vitamin content depend on the food of the mother; any deficiency in her diet will be reflected in the diet of the infant. It is admittedly by no means rare to find well-established rickets in a breast-fed infant. They make the bold suggestion that in many cases a good brand of dried milk may be better for the infant than breast milk of the quality it is receiving. Inquiry into deaths due to causes other than respiratory disease seemed to show that most of the marasmus and some of the enteritis and prematurity cases occurred where the environment was bad, and that often the parents of those infants appeared to be of poor stock, with little physical or mental ability. The investigators suggest a series of simple experiments on infant and maternal feeding which might be carried out at welfare centres, to decide how far the customary diet of pregnant and nursing women is complete in those materials essential to the child.

FRIEDMANN'S TURTLE VACCINE.

EVER since its first appearance in Germany the Friedmann turtle tubercle vaccine [1] has been a trial to the serious worker in the immunological field. It has produced a large mass of clinical records, called forth at least one government report, stimulated the appearance of a competing vaccine made from a rival strain (the Piorkowsky bacillus), and finally produced a lawsuit. But in spite of all this publicity, inquiries into the purely biological properties of the bacillus have never been as thorough as they might have been. M. A. Saenz [2] has at last removed this reproach by making—we hope once and for all—a detailed investigation. The organism was first isolated by Friedmann in 1903 from the lungs of two sea-turtles captured off Italy and removed to the Berlin aquarium. Since its isolation it has undergone many vicissitudes and, as always with a strain passed from laboratory to laboratory, the purity of culture has frequently been called in question. Sea-turtles with pulmonary tuberculosis do not come the way of bacteriologists every day in the week, and laboratories have been unable, therefore, to check the original findings. Saenz has made a careful study of three cultures of the bacillus which he has obtained, and in the *Annales* of the Pasteur Institute sets forth in full detail the morphological characters, cultural properties, and pathogenic action of the organism.

[1] See report of discussion by Tuberculosis Association, THE LANCET, 1929, ii., 36.

[2] Bull. Acad. de Med., 1931, cv., 853; Ann. Inst. Pasteur, July, 1931, p. 4.

He concludes that the pathogenic action of the turtle bacillus for the usual laboratory animals is limited to benign local lesions analogous to those produced by strains of saprophytic acid-fast bacilli. A tuberculin prepared from the bacillus was of very weak toxicity, and an antigen prepared after the manner of Boquet and Nègre showed only the properties of other antigens of acid-fast saprophytes. A vaccine made from the bacillus possessed neither prophylactic nor curative properties when tested in laboratory animals. This effect of the vaccine is even more discouraging than the clinical results reported a year ago by W. C. Fowler [3] in a short series of cases at Pinewood Sanatorium, and there seems to be little doubt that the acid-fast saprophyte will not perform the immunising functions of the true tubercle bacillus. Unfortunately, the exponents of the vaccine are not always ready to believe that Friedmann's bacillus is really nothing more than an acid-fast saprophyte.

ROUTINE MEDICAL EXAMINATION OF UNIVERSITY STUDENTS.

IN a communication lately published by the Office International d'Hygiène Publique, an account is given of an experiment in preventive medicine carried out at the University of Strasbourg on the initiative and with the pecuniary aid of the Comité National d'Études Sociales et Politiques. The experiment was begun in May, 1929, and among the 66 first-year medical students examined some were found to be suffering from ailments which had been overlooked. This initial success led to further developments in the scholastic year 1929–30, when quite an elaborate system of examinations was devised, with as many as five different attendances: one for a general examination, including measurements of height, weight, blood pressure, &c.; one for an examination of the bones, muscles, and nervous system; a third for the eyes; a fourth for the ears, nose, and throat; a fifth for the teeth. These examinations, carried out by 16 doctors and one dentist, were timed so as not to interfere with the classes of the students. Of 861 first-year students, 406 voluntarily undertook to run the gauntlet of all five examinations. It was found, however, that the attendances at the last three examinations began to fall off, several of the students finding that the first two clinical examinations satisfied their requirements. This experience has led to the reduction of the number of attendances or examinations from five to two, and to putting the special examinations before the general clinical examination. The tabulated findings obtained by this system of combing out are already quite impressive. There were seven cases of pulmonary tuberculosis, five of which had hitherto been overlooked, and 77 cases of cicatricial lesions of the pleura, lungs, or bronchial glands, in 22 of which future periodic supervision was considered necessary. There were eight cases of organic and seven cases of functional disease of the heart requiring special precautions, and eight cases of appendicitis, most of them discovered for the first time. There were also as many as six cases of hyperthyroidism. The ophthalmic examination revealed errors of refraction in 136 cases, in 41 of which they had been overlooked or badly corrected. The tonsils and nasopharynx were found to be in a septic state in 23 students, and among the 259 who submitted themselves to a dental examination there were as many as 198 with neglected dental ailments.

It is probable, we learn, that the example of Strasbourg will soon be followed by the Cité Universitaire of Paris.

[3] Tubercle, 1930, xii., 12; see also THE LANCET, 1930, i., 807.

THE VIRUS OF MUMPS.

As long ago as 1908 S. Granata produced evidence that the causal agent of mumps belonged to the group of filtrable viruses, and since that time the investigations of C. Nicolle and E. Conseil (1913), M. H. Gordon [1] and M. Wollstein [2] have confirmed and extended this observation. It is true that serial passage in experimental animals was difficult to obtain; in fact, Wollstein alone was successful in this direction, and even she found that the virus soon diminished in virulence and eventually died out. Nevertheless these investigations had this in their favour, that the experimental disease resembled the human one. The more recent claim of Y. Kermorgant,[3] that the virus of mumps is a spirochæte with a filtrable phase, does not carry conviction, and has not been confirmed. F. F. Tang [4] now produces evidence of having obtained yet another virus from mumps. This came, apparently, from the saliva of an adult on the third day of disease, and was capable of passing through a Berkefeld V. candle. It was pathogenic for the guinea-pig, but not for the rabbit or cat—a fact not in keeping with the work of earlier investigators. But the dissimilarity of Tang's work and those of his predecessors in this field does not end there. The disease produced in guinea-pigs by his virus bore no resemblance to mumps. It was characterised by hæmorrhages in the subcutaneous tissues and muscles of the extremities, particularly about the joints, in the lungs, and around the costochondral junctions; and it was highly lethal, for the guinea-pig at any rate, when first isolated. Tang further admits that about the time when these transmission experiments were being made an epizoötic broke out amongst his normal guinea-pigs, the pathological picture of which bore a striking resemblance to that produced by his mumps virus. He was, however, unable to transmit this spontaneous disease, and he got some evidence that the condition might be due to an organism resembling *Bacillus bronchisepticus*. Despite this, it would appear more than probable that Tang was dealing with an extraneous virus, and not the virus of mumps.

[1] THE LANCET, 1927, i., 652.

[2] Jour. Exp. Med., 1916, xxiii., 353; 1918, xxviii., 377; 1931, xxxiv., 537.

[3] Ann. Inst. Pasteur, 1925, xxxix., 565.

[4] Nat. Med. Journ. China, 1931, xvii., 360.

BIRTHS AND DEATHS IN SOUTH AUSTRALIA.

IN many respects the latest returns from the State of South Australia [5] show vital indices with which western Europe is familiar—for example, a birth-rate declining more rapidly than the death-rate, a corresponding decline in the rate of natural increase of population, and considerable reduction in the mortality from tuberculosis, but an increased loss from cancer. Both the birth-rate (17·2 per 1000) and the crude death-rate (8·4 per 1000) were in 1930 the lowest in the records of the State. For many years South Australia, in common with the other

[5] Statistical Register of the State of South Australia, 1930-31. Part II. Population and Vital. Compiled from official records by W. C. Johnston, F.S.S., Government Statist and Registrar-General of Births, &c.

States of the Commonwealth and New Zealand, has had a very low mortality-rate for infants under one year of age, compared with other countries. In 1930 it was as low as 48 per 1000 live births. The annual mean death-rate of infants under one year of age for the five years 1895–1899 was 110, and for the five years 1925–1929 only 47—a fall of 63. Almost the whole of this improvement is shown to have taken place at ages above one month and under 12 months. The death-rates under one month, under one week, and under one day of age show very little progress and the great majority of those dying under one month of age died from prenatal causes. These facts are, again, comparable with those of this country, and indicate the direction in which efforts for the saving of infant life should be directed. The Mothers' and Babies' Health Association, of which there are 44 branches in the State, is endeavouring to improve the position.

Over half the population are resident in the "metropolis," defined as an area approximately within a ten-mile radius of the General Post Office, Adelaide, and roughly equal proportions of the working population are employed in agricultural and industrial occupations.

PENETRATION OF DRUGS INTO THE TUBERCLE.

IT has long been held that the tubercle is so avascular a structure as to be inaccessible, at all events in its caseating centre, to substances injected into the circulating blood. Intra vitam staining methods of investigation are however throwing doubt on this conviction. In a series of articles which appeared in the *Journal of Experimental Medicine*, and are now reprinted,[1] Dr. V. and Dr. M. F. Menkin show that a dye injected intravenously rapidly enters the inflamed area and is fixed there so that it fails to reach the regional lymph-node. By means of a colorimetric comparator, the authors have been able to study and record inflammatory reactions as produced in the peritoneal cavity of frogs which had been injected intra vitam with dyes after having had a sterile inflammation induced with aleuronat or turpentine. It was found that the rate of fall of concentration of the dye was almost twice as great in the capillaries of the inflamed as in those of the normal mesentery. In other words, there was an increased passage of the dye through the capillary wall in the inflamed area. Continuing these researches Dr. V. Menkin found that colloidal iron or ferric chloride, when injected into the inflamed peritoneal cavity, was fixed in the cavity and failed to reach the retrosternal lymphatic nodes, whereas, in the absence of inflammation, the iron accumulated in the nodes. These lymphatic nodes in animals with normal peritoneal cavities contained approximately 56 per cent. more iron than did the nodes of animals with inflamed cavities. He further demonstrated that ferric chloride injected into the circulating blood entered an inflamed area in the skin and that the amount of iron in the inflamed areas was much greater than that found in inflamed areas of animals that had received no iron. Dr. Menkin also found that horse serum injected into an inflamed peritoneal cavity penetrated into the blood stream less rapidly than when injected into a normal cavity. Such accumulation of foreign protein, he believes, explains the phenomenon of local anaphylaxis described by Auer in rabbits sensitised to horse serum. For instance, the antigen accumulating in the inflamed ear reacts with antibody, intensifying a pre-existing inflammatory reaction and finally producing necrosis of the ear. In the third article Dr. Menkin records some results of injecting iron into the circulating blood stream in tuberculosis. Rabbits with extensive bovine tuberculosis were given daily intravenous injections of a 0·25 per cent. solution of ferric chloride. Using the Prussian blue reaction, the author found that the blue colour became intense in the caseous or central part of the tubercle. This result however appeared to depend on the activity of the circulation, for the dye which penetrated from the blood stream into the periphery of an inflamed area failed to enter a central part in which the circulation was relatively inactive. Further work is being done to determine the effect of the accumulation of iron in tuberculous areas on the course of development of the disease. Its bearing upon the clinical value of chemotherapy is obvious.

[1] Studies on Inflammation, II.–IV. Twenty-second Report of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis. Philadelphia: Henry Phipps Institute.

LADY OSLER.

A BRIEF memoir of Lady Osler, whose companionship of her famous husband made of her a famous woman, has been written by Mr. Arnold Muirhead, for private circulation. Grace Revere was of Huguenot blood; the Rivoires having fled from France to Guernsey on the revocation of the Edict of Nantes, one of them migrated to Boston and became the ancestor of Paul Revere, the patriot-metallurgist. This was Lady Osler's great-grandfather. She married, when little over twenty years of age, Dr. Samuel Gross, a Philadelphia physician, and much her senior, and their house soon became a centre of hospitality, foreshadowing the famous "Open Arms" at Oxford thirty years later. The marriage lasted thirteen years only, and when Dr. Gross died his widow was still a young woman. In 1892 she married William Osler, a devoted friend of her first husband, and it was at her instance that Osler decided to accept the chair of medicine at Oxford in 1905, after holding for 13 years the professorship at Baltimore. How she helped him in his new career has become a legend illustrative of conjugal harmony, and Mr. Muirhead's notes make us understand the wide affection which she inspired in Osler's enormous circle of friends. The battlefield deprived them of their only son, and grief contributed as much as overwork to William Osler's death not long after the struggle had ceased. But Lady Osler continued for nine years to distribute the hospitality of 13, Norham-gardens (The Open Arms), to retain the affections of old friends and gain that of new ones, and fill her days in discharge of duties to which the memory of William Osler called her. This unpretentious little memoir will bring pleasure to many.

PLACENTAL HORMONE AND UTERINE HÆMORRHAGE.

THE effects of injection of the "anterior pituitary-like hormone" of the human placenta in cases of menorrhagia and metrorrhagia are reported by A. D. Campbell and J. D. Collip.[1] Forty cases were treated with an extract containing 40-day rat units per c.cm., and the dosage employed varied from 1–2 c.cm., injected subcutaneously daily or every other day, over different periods. From their results the authors gained the impression that in menorrhagia of the simpler types treatment for one week before

[1] Canad. Med. Assoc. Jour., July, 1931.

the menstrual period materially reduces the flow, but more severe cases may require treatment for three months or longer before normal periods are established. In metrorrhagia, when the continuous uterine bleeding had been in a measure controlled, the same tendency to the establishment of normal cycles was not observed; also, intervals of amenorrhœa were apt to appear in such cases. Severe pain occurring during a period subsequent to the institution of treatment may be of favourable prognostic value, and it was noted that the duration of treatment was sometimes considerably shortened in cases where gentle curettage had recently been performed. The histological picture of the endometrium obtained by curettage does not necessarily reflect the clinical symptoms. Since the action of this and other hormones is not fully understood, the exact manner in which an active principle of placental origin may restore normal function in a case of excessive menstrual bleeding cannot at present be explained. In certain of the patients treated, cycles dated from a period of metrostaxis of one to five days' duration, occurring 10 to 18 days after the onset of the previous period of excessive uterine bleeding. No enlargement or engorgement of the breast was observed in any of the cases under treatment.

INDUSTRIAL MEDICINE AT GENEVA.

THE sixth International Congress on Industrial Accidents and Diseases, which met at Geneva in the first week of August, was a reunion of the two sections which on the last occasion met, one at Buda-Pesth, the other at Lyons. In future the Congress will combine the two branches of the subject; the next will be held in 1935 at Brussels. One of the best group of papers dealt with skin affections due to work. This subject was introduced by Dr. Prosser White, who put forward certain basic principles determining the action of skin irritants which he classified as: (1) direct, injuring by physico-chemical activity; (2) secondary, causing inflammation by stimulating sensory nerves; and (3) indirect, setting up allergy in sensitive persons. He was followed by Prof. M. Oppenheim (Vienna), who exhibited a fine series of illustrations, and Prof. F. Koelsch (Munich), who mainly considered the compensation aspect of trade dermatitis. Other subjects dealt with were the influence of previous health on accidents and industrial diseases, injuries of the blood-vessels, dust of cement and artificial stones, the reaction of the body to industrial poisons, activity and fatigue. Contributions from France, Belgium, and Germany confirmed the comparatively harmless nature of cement dust, previously claimed by American and English observers. A summary of our knowledge on industrial fatigue was communicated by Dr. H. M. Vernon, who presented the influence of occupational environment as elucidated by the Industrial Health Research Board. The papers contributed to the Congress are to be published in full later in the year. The principle of choosing subjects for discussion was adopted at Geneva. Owing however to the acceptance of a number of independent communications, only the reporters had time to summarise their papers, and discussion had to be omitted. In future it is hoped that the chief papers will be circulated beforehand and taken as read, and that independent papers, if accepted at all, will also be taken as read. The sessions could then be devoted to useful discussions and value obtained from the coming together of experts from all over the world. Something is wrong when authorities from Australia, South Africa, the United States, Great Britain, France, Germany, Austria, Italy, Czecho-Slovakia, and other countries are present, but have no chance of exchanging views in open conclave, even though skilled interpreters are at hand to help. The undoubted success of the gathering must be attributed to personal meetings and conversations which took place outside the lecture halls.

COCKROACHES ON SHIPBOARD.

UNLESS they have voyaged in the tropics, and in wooden ships, few realise how disgusting a pest coakroaches can be—and still are, for Surgeon C. L. Williams of the U.S. Public Health Service tells us[1] that 20,000 to 50,000 cockroaches are not an unusual find in a forecastle after fumigation. Iron and steel shipbuilding has reduced their harbourage, and routine anti-rat fumigation has diminished their numbers, but their complete destruction needs a longer exposure to, or a higher concentration of hydrocyanic acid than that required for rats, if the eggs in their protecting egg-cases are to be killed. Rats succumb to 60 g. (2 oz.) of HCN solution per 1000 cub. ft. of air space, acting for two hours, but five times the quantity is needed to kill all cockroaches in the time. This concentration would be too expensive to use over the whole ship, but might be employed in the inhabited part—superstructure, pantries, galleys, and cabins—costing only an extra £2 or so. This is now done at New York on what might be called moral grounds. Immediately after the anti-rat fumigation of a ship the cockroaches are noticed to be far fewer, but in a week or two they are as many as ever, which leads the ship's population to suspect that the effect on rats is just as transient. If the cockroaches were exterminated at the same time there would be less grumbling about the rat fumigation which, whatever people say, has got to be done. A valuable section of Surgeon Williams's paper emphasises the need for pasting strips of paper over openings above or below cabin doors, and over all cracks found, in order to prevent the loss of so easily diffusible a gas as hydrocyanic acid during the period of fumigation; otherwise half of it may be wasted. With the use of a higher percentage of so dangerous a gas the subsequent ventilation of the ship requires greater care. Chloropicrin, the warning gas which is sometimes added to increase safety, is not more deadly to cockroaches, although they do not, as was once feared, take advantage of the warning to secure more effectual cover. All seafarers will be grateful to Surgeon Williams for information in helping them to get quit of a pest.

[1] Public Health Reports, Washington, July 17th, p. 1680.

POOR-LAW HOSPITALS AND SWEEPSTAKES.—Our Dublin correspondent informs us that a circular letter has been issued by the Department of Local Government and Public Health to the authorities in control of rate-supported hospitals, dealing with their position in regard to sweepstakes. It is stated that one section of the Act imposes on the Minister the duty of applying money received from sweepstakes toward the provision, improvement, or equipment of institutions for the prevention, treatment, or cure of physical or mental diseases, or injuries to human beings. Moreover, it is expressly provided that money so applied should be in addition to, and not in substitution for, or toward, ordinary maintenance, and should not be taken into consideration by the local authority when determining the amount to be expended by them. Decisions with regard to the application of the money cannot be made until the exact amount to be distributed is known, but in the meantime the Minister invites local authorities that intend to provide, improve, or equip an institution and apply for a grant out of the money to be paid to the Minister, to take the matter into consideration and transmit their proposals to the Department.

SPECIAL ARTICLES

EXPERIMENTAL OBSERVATIONS ON DERMATITIS DUE TO DYED FUR.

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IN the winter of 1922–23 it became apparent that the incidence of cases of dermatitis due to the wearing of dyed fur had suddenly increased. Before that period dyed fur had been recognised as a cause of dermatitis, both among those engaged in the preparation of such articles and among those wearing them, but, nevertheless, examples of this type of eruption were rarely seen. The increase noted in 1922–23 was maintained during the winter of 1924, and during these years several papers dealing with the subject were published. In 1924 the Ministry of Health conducted an inquiry into the occurrence of dermatitis attributed to the wearing of fur collars. The main conclusions arrived at in the report of the Ministry[1] (which were based on facts obtained during the inquiry and on papers dealing with the subject in the current medical journals) were as follows: Personal idiosyncrasy on the part of the patient was responsible to some extent for the appearance of the dermatitis, but this susceptibility was specific for a certain class of fur, and did not explain the sudden and unprecedented incidence of fur dermatitis cases. The dermatitis was apparently produced by products of the incomplete oxidation of para- and meta-phenylenediamine. In some instances the mordant used was also thought to be partly responsible. The part played by personal idiosyncrasy was obvious when the proportion of sufferers from fur dermatitis to wearers of dyed furs was considered. The facts recorded in the inquiry did not, however, provide any conclusive evidence as to the nature of the actual constituent in the fur which was responsible for the dermatitis, and any statements regarding this important point were merely tentative. During the process of dyeing, a fur may be treated with various chemicals, including the actual dye itself, and any one of these is theoretically a potential skin irritant. The fact that a particular or even chemically unidentifiable oxidation product of an aniline dye can be isolated from a fur which is known to have been treated with the mother substance of the isolated compound is no proof that such a compound is the cause of the dermatitis which the fur has produced. Direct proof of the specific irritant properties of a substance on the human skin can only be obtained from biological experiments with the substance in question. A more precise knowledge of the nature of the specific susceptibility of the skin towards the irritant may also be obtained if such methods are employed in the investigation of the problem.

Cases of dermatitis due to dyed fur, although less numerous than in the years 1922–24, are still frequently met with. Table I. shows the total number of such cases and their percentage of the total number of cases of dermatitis in females over 15 years of age who have been treated in the skin department of the Edinburgh Royal Infirmary during the decennium 1920–30:—

TABLE I.

A = Total number of cases of dermatitis in females over 15.
B = Number of cases due to fur.
C = Percentage of total due to fur.

Year.	A.	B.	C.	Year.	A.	B.	C.
1920	174	1	0·57	1926	238	16	6·72
1921	185	1	0·54	1927	168	7	4·18
1922	135	1	0·73	1928	252	13	5·15
1923	194	22	11·34	1929	362	23	6·35
1924	233	37	15·88	1930	385	13	3·37
1925	201	17	8·45				

For obvious reasons, the complaint is almost exclusively met with in females, so that figures dealing only with dermatitis in females have been given for the total annual number of dermatitis cases in the Table. As no cases of fur dermatitis have been met with in young children, the numbers given for all classes of dermatitis have been limited to cases over 15 years of age. The number of cases of fur dermatitis given for 1920–22 may be slightly low, since attention was not concentrated on the possibility of fur being the cause of dermatitis until the condition became prevalent in 1923. Nevertheless, the abrupt rise in 1923–24 is undoubtedly real, for the numbers were then much higher than in the succeeding years, during which the condition was well recognised.

Since the incidence of this type of eruption is still considerable, an investigation into its causation and nature has been undertaken. Furthermore, as the problem involved was a concise one, dealing with a type of dermatitis due to an irritant which could presumably be definitely ascertained, it was thought that any observations which might be made during the course of the investigation would have some bearing on the related problems presented by other types of dermatitis, either due to external or internal causes.

Investigation.

The irritant properties of two lambskin furs, one dyed brown and the other grey, which were known to have caused dermatitis, were examined. The experiments carried out with the brown fur were of a preliminary nature to test out the methods which it was proposed to use throughout the investigation, while those performed with the grey fur were more detailed, and were pursued further.

The main issues of the problem may be stated as follows:—

(1) Is the skin irritation produced by a dyed fur due to the fur itself or to the chemicals used in the dyeing process?

(2) Are any special conditions, such as moisture or a greasy skin, necessary before a dyed fur can produce skin irritation, and, if so, will apparently normal skins react to certain dyed furs provided such favourable conditions are present?

(3) Can a skin, which has previously tolerated a given fur, subsequently develop an eruption when again brought into contact with this fur?

(4) What substance in the fur is responsible for the production of dermatitis?

(5) What concentration of this substance is required in order to produce dermatitis?

(6) Does the action of the irritant substance, when this is present in the concentration required to produce dermatitis, differ on normal skins and on the

* In receipt of a part-time grant from the Medical Research Council.

skin of subjects who have suffered from dermatitis due to the particular irritant in question? In other words, is the irritation which is produced by the substance entirely due to a constant specific pharmacological action depending on and proportional to its concentration, or must the skin possess or develop the property of reacting to the application of the substance with a special type of reaction before the latter can produce any harmful effects?

(7) If such a qualitative difference does exist between normal skins and those which are irritated by compounds contained in dyed fur, what is the degree of this difference?

(8) Can the irritant properties exhibited by a given dyed fur be removed by appropriate chemical or physical manipulation?

The method used throughout in the investigation of these questions was that of the contact eczema test.

This test was introduced by Jadassohn and Bloch to detect the external irritant in cases of dermatitis, and since its introduction it has been extensively used on the continent as a routine practice in the investigation of such cases, and in special investigations on the nature of dermatitis. The method is simple, and consists in the application of the suspected irritant in low concentration, either in solution, paste, or ointment form, to a small area of the skin of the back. Solutions are applied as soaks on small linen squares. The test application is covered with linen or oiled silk and kept in position with adhesive plaster. The application is removed at the end of 24 hours, and the skin examined. If the test is positive, the skin which has been in contact with the test substance is red and slightly œdematous, and the surface is studded with minute vesicles. It presents the appearance of a localised area of dermatitis. The area in direct contact with the adhesive plaster is frequently red and the hair follicles prominent, but this quickly subsides, although in some cases the skin is actually sensitive to the ingredients of the plaster, and the redness persists. Sometimes a positive reaction is not evident when the tests are removed and the skin examined at the end of 24 hours, but develops during the succeeding 24 hours. It is necessary, therefore, to verify all negative reactions 24 hours after removal of the test applications, in case a late positive reaction develops. A positive result is a direct proof that the substance applied is capable of producing a dermatitis in the concentration employed, and the degree of sensitivity of the skin towards the substance can be ascertained by varying the concentration of the latter.

While such tests are of immense value in elucidating the cause of individual cases of dermatitis, they have also been used to demonstrate the relative susceptibility of the skin of patients suffering from dermatitis to react to several types of irritants, as compared with the skin of normal individuals. Thus Jaeger[2] found that 50 per cent. of cases of dermatitis gave an abnormal inflammatory reaction to certain volatile oils, while only 5 per cent. of normal individuals reacted. Using similar substances, Bloch[3] found that 35 per cent. of the dermatitis cases reacted, and only 5 per cent. of normal individuals gave positive reactions. Evening[4] obtained positive results in 36 per cent. of dermatitis cases, and 2·2 per cent. of normal individuals, and Schürch[5] obtained results of a similar order.

BROWN LAMBSKIN DYED WITH PARAPHENYLENEDIAMINE.

The first fur to be investigated, using this method, was a brown lambskin which had been dyed with paraphenylenediamine. Three female patients, who were known to have suffered from dermatitis of the neck, due to the wearing of dyed fur collars, were tested. In each case the rôle of the collar in the production of the dermatitis had been proved on several occasions by its disappearance when the fur ceased to be worn, and its subsequent reappearance on the reapplication of the fur to the neck. Three tests were made in each of the three cases, using the patient's own fur, the brown lambskin and undyed lambskin. Portions of the furs were clipped off the skins, and three applications were made with each fur, a sample of each fur being applied dry, moistened with distilled water, and moistened with saline. The tests were removed and read at the end of 24 hours, and again at the end of a further 24 hours. The results are given in Table II.

TABLE II.

Results of contact tests with paraphenylenediamine-dyed brown lambskin fur applied for 24 hours in three patients who had suffered from dermatitis due to dyed fur collars.

M.D.W. = Moistened with distilled water.
M.S. = Moistened with saline.

Case.	Patient's own fur.			Dyed brown lambskin.			Control undyed lambskin.		
	Dry.	M.D.W.	M.S.	Dry.	M.D.W.	M.S.	Dry.	M.D.W.	M.S.
1	+ + +	+ + +	+ + +	−	+ + +	+ + +	−	−	−
2	+ + +	+ + +	+ + +	+ +	+ + +	+ + +	−	−	−
3	+	+ + +	+ + +	+	+ + +	+ + +	−	−	−

\+ = redness persisting for 24 hours.
\+ + = redness and œdema.
\+ + + = redness, œdema, and minute vesiculation of the surface, and itching.
\+ + + + = redness, marked œdema, and vesiculation of the surface and itching.

The positive reactions to a single application of the dyed furs persisted for 10 to 15 days, passing through a dry scaly stage before the skin resumed its normal appearance.

It is evident from these results that the irritant properties of the dyed lambskin are due to one or more of the constituents used in the dyeing process. It has also been shown that moistening the fur with either distilled water or saline solution increases its irritant properties, although it is to be noted that preliminary moistening is not necessary for the production of a positive reaction. In all cases the patient's own fur and the dyed brown lambskin fur caused some staining of the skin, even when applied in the dry state, and this staining was due to some of the dye becoming dissolved either in the moistening fluids or in the perspiration or oily secretion of the skin. Case 2 had a coarse, greasy skin, and in her the dry applications caused more staining than in Cases 1 and 3. The fact that the reactions to the dry furs were less marked than those to the moist preparations suggests that the concentration of dissolved dye bears a direct relationship to the intensity of the reaction which it produces.

To control these observations, the brown lambskin fur was applied dry, moistened with distilled water, and with saline, to the skin of 15 patients suffering from various types of skin diseases. The patients were retested with similar applications after an interval of 16 days to verify the results of the first tests and to ascertain whether it was possible for a skin which had shown a negative reaction to the first application to develop an altered reaction as a result of this application, so that a positive reaction would be given to subsequent tests. The results are given in Table III.

The fur did not produce a positive reaction in these patients when applied for the first time. In most of these cases all the fur applications produced some degree of staining of the skin, and in several the skin was excessively greasy. This staining is well shown in Fig. 1. It may therefore be concluded that normal skins do not tend to react to the dye dissolved from the brown lambskin fur, even when this substance is in prolonged contact with the skin, either in watery or oily solution. One case, however, developed a

positive reaction to all three applications of the fur on testing a second time, although the results of the first test had been negative. This patient's skin apparently developed a susceptibility towards some substance in the fur, and presumably the altered reaction which occurred had been brought about as a result of the first application.

TABLE III.

Results of tests with brown lambskin fur in 15 patients suffering from various types of skin disease. The fur was applied dry, moistened with distilled water, and with 0·9 per cent. saline. Each patient was retested after an interval of 16 days.

Disease.	Cases.	First test.		Second test.	
		Pos.	Neg.	Pos.	Neg.
Dermatitis venenata ..	2	0	2	0	2
Dermatitis..	3	0	3	0	3
Dermatitis artefacta ..	2	0	2	0	2
Lupus	1	0	1	0	1
Sycosis	1	0	1	0	1
Impetigo	1	0	1	0	1
Asthma and prurigo ..	1	0	1	0	1
Ulcus cruris	4	0	4	1	3

Pos.=positives; Neg.=negatives.

These preliminary experiments show that the skin of patients who are the subjects of fur dermatitis reacts differently from that of normal individuals to the application of a dyed fur which possesses irritant properties, even when the conditions favourable to the production of a dermatitis reaction are equal in both types of individual. They also show that the

FIG. 1.

This patient's skin was greasy, and marked staining of the skin with the dye from a brown lambskin fur applied dry (left) and moistened (right) has occurred. The dry preparation has caused almost as much staining as the moist. No reaction followed either application.

skin of a patient suffering from dermatitis, due to some other cause, is not specially apt to react to dyed fur, suggesting that the constituent in the dyed fur responsible for the reaction in susceptible cases is not a universal irritant for eczematous individuals. A specific capacity on the part of the skin to react with such substances appears to be a necessary factor before any reaction takes place. This capacity bears no relationship to the oiliness or moisture of the skin, as shown by the negative reactions obtained in normal individuals in which such conditions were present to a marked degree. The experiments further demonstrate that it is apparently possible for a previously normal skin to develop a sensitivity towards dyed fur as a result of one application of the fur.

EXPERIMENTS WITH GREY LAMBSKIN.

On the basis of these preliminary experiments, a further series of experiments was carried out to test the irritant properties of a grey dyed lambskin, which had been responsible for a few cases of dermatitis. This fur had undergone treatment with solutions of proprietary substances, which will be referred to as A, B, C, and D. Substance A, which belonged to that class of phenylenediamine compounds known as ursols, and which contained paraphenylenediamine, but not metaphenylenediamine, gave a purplish-grey solution when allowed to oxidise in air for several days, and when mixed with hydrogen peroxide developed a medium-grey colour. Solutions of the other substances were colourless. In the dyeing process hydrogen peroxide was used to develop the required grey colour.

Contact tests were performed with this fur in 50 patients suffering from various types of skin lesions, but with no history of previous fur dermatitis. The fur was applied moistened with distilled water, since in the preliminary experiments with the brown lambskin fur it had been shown that in fur-sensitive individuals a more intense reaction was given when the fur in question was applied moist. Each individual in the series was retested with the fur after an interval of 16 days had elapsed from the time of the first test. The results are shown in Table IV.

TABLE IV.

Results of contact tests with moistened dyed grey lambskin in 50 patients suffering from various skin lesions. The tests were repeated after an interval of 16 days.

Disease.	Cases.	First test.		Second test.	
		Pos.	Neg.	Pos.	Neg.
Dermatitis	22	0	22	0	22
Psoriasis	7	0	7	0	7
Ulcus cruris	4	0	4	0	4
Nævus	3	0	3	0	3
Lupus vulgaris	3	0	3	0	3
,, erythematosus ..	2	0	2	0	2
Impetigo contagiosa ..	2	0	2	0	2
Sycosis	2	0	2	0	2
Prurigo	2	0	2	0	2
Xeroderma	1	0	1	0	1
Lichen planus	1	0	1	0	1
D. herpetiformis	1	0	1	0	1
Total	50	0	50	0	50

The results show that the fur does not contain any substance which has universally toxic properties in the concentrations obtained in the experiment. Again, staining of the skin by the fur was frequently in evidence.

Twelve patients known to have suffered previously from fur dermatitis were then tested with moist applications of the grey dyed lambskin fur. The clinical histories of those patients strongly suggested that the dermatitis from which they had suffered was due to the wearing of dyed fur; the dermatitis persisted until the fur ceased to be worn, and eczema tests with the fur afforded absolute proof of the causal relationship of the fur to the dermatitis. In some cases other types of dyed fur, which were substituted for the originally offending article, also caused dermatitis, while in others a dyed fur had been found which could be worn with impunity.

Each of these patients gave a positive reaction to the test application of the grey lambskin. In four cases the positive reaction was not evident until 24 hours after the test applications had been removed—i.e., 48 hours after their first application. The reaction was intense in every case, and consisted of redness, œdema, and minute vesiculation, and was accompanied by a considerable degree of itching. From the clinical histories of these patients it will be seen that, although the patient had suffered from dermatitis produced by a fur collar, it had been

subsequently possible to obtain and to wear a dyed fur which did not produce any harmful results. This being so, the results of the tests with the grey fur might well have been negative.

The positive results obtained in these cases, when compared with the uniformly negative results found in other types of skin disease, demonstrate the existence of a special reactivity or sensitivity on the part of the skin in the former group. They also show that patients who have shown this type of sensitivity to one particular dyed fur are extremely liable to react similarly to other types of dyed furs.

Since several chemical substances had been employed in the preparation of this fur, these were tested out separately on fur dermatitis Cases 1, 2, 4, 9, and 13, and also on 50 patients suffering from various types of skin disease. The following test solutions were used :—

Solution I. = 0·5 per cent. aqueous solution of "A."
" II. = 0·5 " " " "B."
" III. = 0·5 " " " "C."
" IV. = 0·5 " " " "D."

The results in fur dermatitis cases are given in Table V.

TABLE V.

Results of contact tests with solutions I. to IV. applied for 24 hours in fur dermatitis cases.

Case.	Solution.			
	1	2	3	4
1 ..	+ + + +	—	—	—
2 ..	+ + + +	—	—	—
4 ..	+ + + +	—	—	—
9 ..	+ + + +	—	—	—
10 ..	+ + + +	—	—	—

Forty-nine of the 50 cases suffering from skin disease, other than fur dermatitis, gave a negative reaction to all four test solutions on the first application, and also on a second application performed after an interval of 16 days. One case gave a strong positive reaction to solution 1 (0·5 per cent. "A"), while the reactions to solutions 2, 3, and 4 were negative. This patient was suffering from a leg ulcer, and had never worn dyed fur. When tested with the grey dyed lambskin she gave a strongly positive reaction.

The above results definitely prove that the potential irritant properties possessed by the grey lambskin fur are due exclusively to the presence therein of "A," or of some decomposition or oxidation product of this substance which had developed in the 24 hours during which it was in contact with the skin. The essential part played by a specific sensitivity of the skin in the production of dermatitis has again been demonstrated, and the results obtained with the fur itself have been confirmed and more accurately defined. The specific sensitivity of the skin which is associated with the dermatitis reaction is not possessed to any extent by normal skins.

In all, 100 cases of various types of skin disease (other than fur dermatitis cases) were tested either with the grey fur or with "A," and of these only one displayed any evidence of sensitivity, whereas a positive reaction was given by all fur dermatitis cases tested. Such a reaction must, therefore, be considered as abnormal, and must depend on some defect in the skin itself.

In order to ascertain whether it was unaltered "A" or its oxidation products which were reacting with the abnormal skin, a 24-hour old mixture of 0·5 per cent. "A" solution and hydrogen peroxide was tested on fur dermatitis Cases 1, 2, 4, 9, and 10. The results are given in Table VI.

These observations show that the positive results obtained with the fur itself and with freshly prepared 0·5 per cent. "A" solution were due to the skin reacting either with unchanged "A" or with its early oxidation products, and that the more completely substance "A" was oxidised the less was the sensitised skin likely to react with it.

TABLE VI.

Results of contact tests with 0·5 per cent. solution of compound "A" mixed with hydrogen peroxide for 24 hours.

Case	1	2	4	9	10
0·5 per cent "A" solution + hydrogen peroxide	—	—	+ +	—	+ +

While the marked contrast between the number of positive reactions obtained in fur dermatitis cases and in non-fur dermatitis cases is strong evidence that, in the former group, the skin is hypersensitive to substance "A," it gives no indication of the degree of this hypersensitivity. The following experiments were performed in order to gain an approximate quantitative estimate of this factor. Fifty patients suffering from various types of skin disease who had previously given a negative reaction either to 0·5 per cent. "A" or to the grey lambskin fur were tested with 1 per cent., 5 per cent., and 10 per cent. solutions of "A." No positive reactions were obtained with the 1 per cent. solution, negative reactions were given by 48 patients to 5 per cent. and 10 per cent. solutions, and two cases reacted to 5 per cent. and 10 per cent. solutions. Fur dermatitis Cases 1, 4, 5, 10, 11, and 12, and the case of leg ulcer, which had given a positive reaction to 0·5 per cent. "A" and to the grey fur (Case 13) were tested with 0·5 per cent., 0·1 per cent., 0·05 per cent., and 0·005 per cent. solutions of "A" and with the grey fur. The results are shown in Table VII.

TABLE VII.

Results of contact tests with grey lambskin fur, 0·5 per cent., 0·1 per cent., 0·05 per cent., and 0·005 per cent. solutions of "A" in fur dermatitis and "A" sensitive cases.

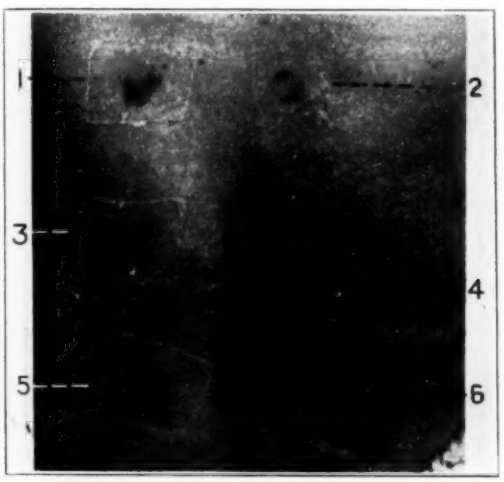
Cases.	Fur.	Solutions of "A" (per cent.).			
		0·5	0·1	0·05	0·005
1	+ + + +	+ + + +	+ + +	+ + +	—
4	+ + + +	+ + + +	+ + +	+ + +	+
5	+ + + +	+ + + +	+ + +	—	—
10	+ + + +	+ + + +	+ + +	—	—
11	+ + + +	+ + + +	+ + +	—	—
12	+ + + +	+ + + +	—	—	—
13	+ + + +	+ + + +	+ + + +	+ + + +	+ + + +

These experiments show that the difference in sensitivity to dyed fur between a skin which is intolerant to it and a normal skin is of considerable magnitude. Thus Cases 4 and 13 react to at least 1/2000 of the concentration of "A" to which a normal skin is tolerant. Case 4, Cases 5, 10, and 11, and Case 12 react, respectively, to 1/200, 1/100, and 1/20 of the same concentration. The position might be regarded from the opposite standpoint, and Cases 4 and 13 considered as being at least 2000 times, Case 4 200 times, Cases 5, 10, and 11 100 times, and Case 12 20 times as sensitive to "A" as the normal skin. Moreover, it is possible that the normal skin would tolerate concentrations of "A"

higher than 1 : 10, in which case the sensitivity of the reacting skins would be correspondingly greater.

When the positive reactions obtained with varying dilutions of "A" are compared with those obtained with the ursol-dyed fur, it is possible to gain some idea of the concentration of "A," which is presumably dissolved out of the fur and brought into contact with the skin to give rise to the positive skin reaction produced by the fur. In Cases 1 and 2 this must have been between 0·05 per cent. and 0·005 per cent.; in Cases 5, 10, and 11 between 0·05 per cent. and 0·1 per cent.; and in Case 12 between 0·5 per cent. and 0·1 per cent. Case 12 further demonstrates that it is possible for a solution of "A," having a concentration of at least between 0·1 per cent. and 0·5 per cent., to be formed from the grey fur when this becomes moist.

FIG. 2.



Case 6, Table VIII., tested with fur samples 1–6.

					After application of test—
1	=	Reaction to fur	3	.....	36 hours.
2	=	,, ,, ,,	2	.....	36 ,,
3	=	,, ,, ,,	1	.....	36 ,,
4	=	,, ,, ,,	4	.....	36 ,,
5	=	,, ,, ,,	5	.....	36 ,,
6	=	,, ,, ,,	6	.....	36 ,,

An interesting and important point, conclusively shown by the results of the tests with different concentrations of "A," is that the sensitivity of the skin in fur-sensitive cases shows wide individual variations. The results also show, although less definitely, that the intensity of the reaction in individual cases varies directly with the concentration of "A." It seems probable, however, that the range of concentrations throughout which graded reactions occur is small, and that, once a certain concentration is reached, the intensity of the reaction obtained in maximal.

THE EFFECTS OF CLEANING.

The substance in the grey fur which took part in the production of the reaction exhibited by abnormal skins when in contact with the fur having been identified as substance "A" or an early oxidation product of this substance, attempts were made to ascertain whether it could be removed by special methods. For this purpose seven samples of lambskin fur dyed with "A," but subjected to different cleaning processes, were tested on eight fur-sensitive cases, along with the original grey lambskin used in the previous experiments. The cleansing processes consisted in washing in water or benzene for varying periods, brushing, or drumming with sand. It was not possible to test out all eight samples in each case, while in some individual cases the same sample was tested on several occasions. The results are given in Table VIII.

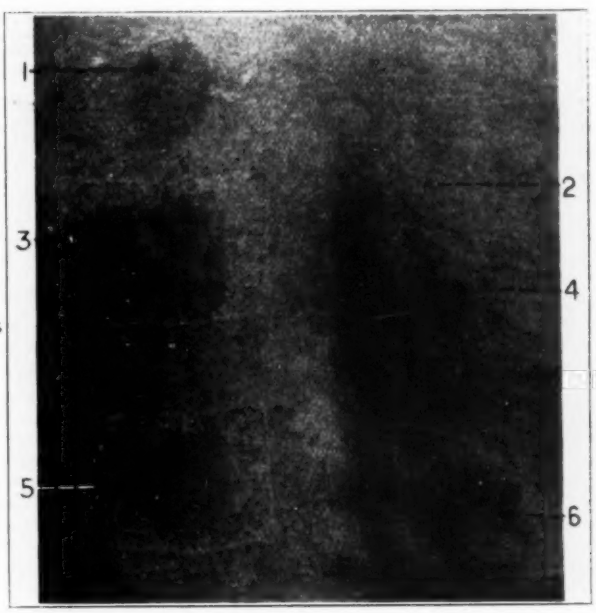
TABLE VIII.

Results of contact tests with original "A" dyed grey fur (fur 1) and with seven grey furs dyed with compound "A," and subjected to various cleansing processes, in fur-sensitive cases.

Case.	Fur. Sample. 1	2	3	4	5	6	7	8
1	+ + + +	0	0	0	0	–	+ + +	+ + +
2	+ + + +	0	0	0	0	–	+ + +	+ + +
4	+ + + +	+ +	+ +	+ +	+ +	–	+ + +	+ + +
5	+ + + –	+ +	0	+ +	0	–	+ + +	+ + + +
*6	+ + + +	+ +	+ +	+ +	+ +	–	+ + +	+ + + +
7	+ + + +	+ +	+ +	+ +	–	–	+ + +	+ + + +
8	+ + + +	+ + +	+ + +	+ + +	+ +	–	+ + +	+ + + +

* Reactions to samples 1–6 shown in figure (2). 0 No test performed.

FIG. 3.



Reactions given by Case 4 to samples 1, 2, 3, 4, and 5.

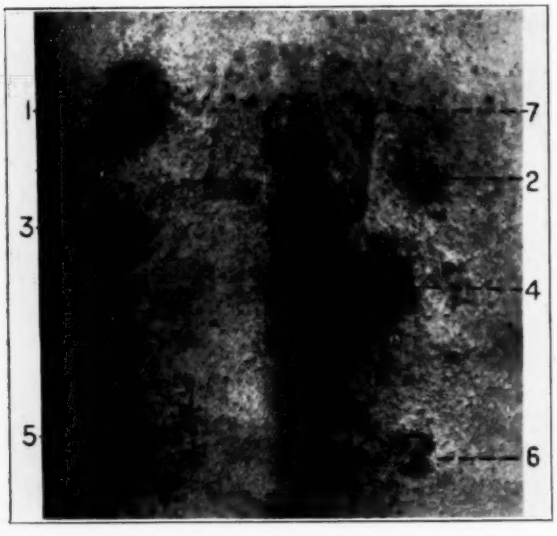
					After application of test—
1	=	Reaction to fur	1	.....	5 days.
2	=	,, ,, ,,	5	.....	5 days.
3	=	,, ,, ,,	1	.....	30 hours.
4	=	,, ,, ,,	2	.....	30 ,,
5	=	,, ,, ,,	4	.....	30 ,,
6	=	,, ,, ,,	5	.....	30 ,,

This Table shows that with adequate cleaning methods it is possible to reduce considerably, and perhaps to eliminate altogether, the substance in the dyed fur which causes the hypersensitive skin

to give a dermatitis reaction. A comparison with the reactions given by Case 4 in Tables VI. and VII. shows that any solution of "A" derived from fur 6 when in contact with the skin must have been of a lower concentration than 0·005 per cent., since Case 4 was capable of reacting to this concentration, whereas she gave no reaction to fur 6.

In this investigation the reactions given by Case 4 were of special interest. This patient was first tested with furs 1, 3, and 6. No reaction was obtained with fur 6, while a well-marked reaction occurred with fur 1, and a papulo-erythematous vesicular reaction occurred with fur 3. The reaction to fur 1 was still visible at the end of five days as a dry, pink, scaly area, but that to fur 3 had almost faded. At this time fur 1 was reapplied, together with furs 2, 4, and 5; 24 hours later the tests were read and positive results (Fig. 3) were found in each case. During the following few days a flare-up of the subsiding positive reactions to the first application of fur 1 and of fur 3 occurred. This exacerbation gradually subsided, along with the reactions to the second application of fur 1 and of furs 2, 4, and 5 during the succeeding 37 days. Twelve days after the second application of fur 1, and at a time when the reactions to this application and to furs 2, 4, and 5, and also the exacerbation of the reactions to the first application of furs 1 and 3, were subsiding, a third application of fur 1 was made. A well-marked erythemato-vesicular reaction

FIG. 4.

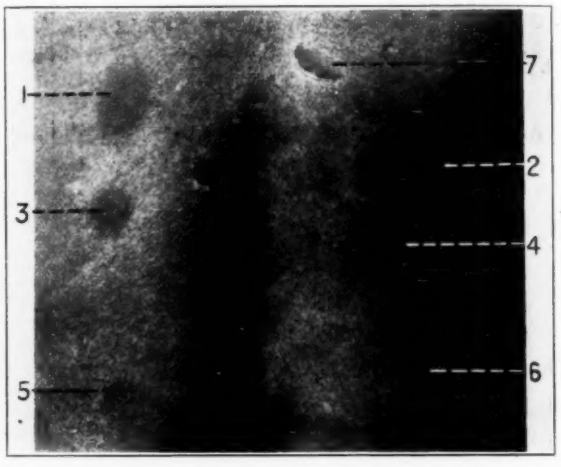


Reaction given by Case 4 to samples 1, 2, 3, 4, and 5.

			After application of test—
1 =	Persisting reaction to fur	1	17 days.
2 =	" " " "	3	17 "
3 =	" " " "	1	12 "
4 =	" " " "	2	12 "
5 =	" " " "	4	12 "
6 =	" " " "	5	12 "
7 =	Reaction to " "	1	30 hours.

followed (Fig. 4), but, contrary to expectations based on the experience with fur 3, there was no general flare-up of the fading reactions to furs 1, 2, 3, 4, and 5. Fifty days from the date of the first test applications the skin of the back had resumed its normal appearance. Fur 3 was again applied at this time, and a positive result was obtained at the site of application only (Fig. 5). The interesting feature of these results is the flare-up

FIG. 5.



Reactions given by Case 4 to samples 1, 2, 3, 4, and 5.

1, 3, 4, 5, 6, 7 represent the corresponding sites in Figs. 2 and 3 50 days after the first tests were applied. The reactions have completely faded, leaving only a faint pigmentation of the skin.

2 = Reaction to fur 3, 36 hours after the application of the test.

of subsiding positive reactions on one occasion, which was coincident with the application of fresh tests to adjacent areas. This point will be discussed later.

ATTEMPTS TO TRANSFER SKIN SENSITIVITY TO NORMAL SKINS.

Serum was obtained from Cases 4 and 13, both of whom had shown the greatest skin hypersensitivity to "A," and 0·2 c.cm. of each sample was injected intradermally into four areas of the skin of the forearm in four individuals who had given a negative reaction to contact tests with 10 per cent. "A." After four hours the injected areas were tested with 0·5 per cent. "A" applied to a scratch and also as a contact test. (Method of Prausnitz and Küstner.) Control scratch and contact tests were made on adjacent uninjected skin. No positive reactions were obtained with any of the tests. Both forms of test were repeated 24 hours later, but again the results were negative.

SUMMARY OF THE FOREGOING EXPERIMENTAL RESULTS.

(1) The potential irritant properties of a dyed fur are due to some substance used in the dyeing process, and not to mechanical irritation by the fur.

(2) Normal skins do not react to a dyed fur even under conditions in which the fur in question would produce dermatitis in a fur dermatitis patient.

(3) The dermatitis is due to a hypersensitive skin reacting with some substance in the dyed fur in concentrations with which normal skins do not react, and, therefore, this reaction may be termed allergic.

(4) The substance in the fur investigated with

which the allergic skin reacts is "A," or an early oxidation product of this compound.

(5) Fur dermatitis cases react to concentrations of "A," varying from 0·005 per cent. to 0·5 per cent., and in these cases there is considerable individual variation with regard to the concentration necessary to produce dermatitis.

(6) Most normal skins fail to react with a 10 per cent. solution of "A" although some were found to react with a 5 per cent. solution.

(7) The degree of sensitivity of the skin to dyed fur found to exist in fur dermatitis cases ranged from at least 200 times to 2000 times that found in non-fur dermatitis cases.

(8) It is possible for at least a 0·5 per cent. solution of "A," or its oxidation products, to be formed in the fluid secretions of the skin when in contact with the dyed fur. This type and concentration of solution does not produce dermatitis in non-fur dermatitis cases.

(9) Dermatitis resulting from a single application of a minute concentration of a chemical towards which the skin is allergic may persist for several weeks.

Comment.

Two conclusions may justifiably be drawn from the results obtained in the foregoing experiments. The first is that "A" is the only factor present in the fur which takes any part in the production of dermatitis. The second, and the more important from the point of view of its general application, is that the dermatitis depends on an allergic state of the skin, and that it is idiosyncratic in nature. The term allergy is here used in its wider sense, according to the definition of Bloch, which is as follows:—

> "Allergy is that state which has as its basis the property of certain groups of cells (organs) of the living organism to react in a specific manner when brought in contact with a substance which is, as far as is known, foreign to the organ or cells; the characteristic of this specific pathologic process lies in the fact that it is caused by the reaction of this exogenous substance with its specific cellular fixed antibody. The basis and essence of allergy is the ability of the living cell to react with the production of specific antibodies to the stimulus of foreign substances, which are therefore called antigens, as well as the fact that the contact of the antigen with its specific cellular fixed antibody causes a disturbance of cellular life which usually results in an inflammatory reaction."

The term idiosyncrasy means that the reactions caused by the exogenous substances in question occur only in a small percentage of individuals. The reason for idiosyncrasy is therefore the presence of the allergic state in those individuals who are idiosyncratic. As a result of allergy, idiosyncratic individuals react to substances which produce no effect in normal individuals. Such substances are, therefore, irritant and toxic for the former class, but perfectly harmless for the latter. This toxic effect, however, is not proportional to the dose or concentration of the substance coming in contact with the cells, but is entirely dependent on the state of these cells. The substance only precipitates a reaction when the cells are in a state of allergy. Such a reaction is not due to the pharmacological action of the substance, since this would be approximately the same in all individuals, and would be proportional to the amount of the substance acting on the cells. It has been shown that allergic phenomena, which were originally considered to be brought about only by antigens of a protein nature, can be caused equally well by crystalline substances.

The results of the tests show conclusively that positive reactions occur only in idiosyncratic individuals, and that they are due to the allergic state of the skin towards "A." In this class of individual the intensity of the reaction bears no constant relationship to the concentration of "A" applied to the skin. In one case a concentration of 0·5 per cent. may be required, while in another 0·005 per cent. will suffice. The uniformly negative results obtained with 0·5 per cent. "A," and the large percentage of negative results obtained with 10 per cent. "A," in individuals who had not suffered from fur dermatitis, show that even in these strengths the pharmacological action of compound "A" cannot be described as irritant. Nevertheless, throughout the range 0·5 per cent. to 10 per cent. several positive reactions were obtained, and these individuals may be considered idiosyncratic in the same way as those sensitive to lower concentrations, but in the former case the allergy is of a less delicate or sensitive nature. It is possible that with concentrations higher than 10 per cent. compound "A" would display universal irritant properties, which would then depend on the pharmacological action of the drug, be proportional to the dose, and independent of the state of the skin in individual cases. When it is considered that reactions were obtained with concentrations from 1/200 to 1/2000, which was tolerated by normal individuals, it is obvious that some factor other than the direct action of "A" must be mainly responsible for the reaction. This factor can only be the altered state of reactivity of the skin cells, or, in other words, allergy. Apart from the evidence derived from a comparison of the active concentrations in reacting individuals and the concentrations tolerated by individuals, the behaviour of the positive reactions which were obtained denotes their allergic nature. The latent period observed between the removal of the test application and the appearance of a positive result, and the tendency for positive reactions to flare-up as a result of subsequent applications of "A," are both typical of allergic phenomena.

The results obtained by Burgess and Usher[6] are comparable to the more general findings in the present investigation. They found that of 200 normal persons tested with a face lotion which had been associated with dermatitis 0·5 per cent. gave a positive reaction, while a reaction was obtained in all patients who had suffered from the dermatitis. They record the interesting observation that in several of these dermatitis patients a positive reaction could only be obtained at the site of the healed dermatitis, so that in those cases the allergy was localised to certain skin areas. No example of regional allergy was found in the present investigation, and in every case the allergic phenomena were elicited over wide areas of skin. Beyond demonstrating the idiosyncratic and allergic nature of the disease, and proving that only one constituent of the lotion was capable of producing it, Burgess and Usher performed no experiments to demonstrate the quantitative nature of the allergy.

The fact that in the present experiments positive reactions were only obtained when "A" was applied to the intact skin, and that uniformly negative results were obtained by the scratch method, points to the allergy being localised in the epidermis. It also points to the necessity of carrying out both types of test when an allergic condition of the skin is suspected.

Attempts to demonstrate the presence of free antibodies in the serum, by the passive transference method of Prausnitz and Küstner, and so to prove the antibody-antigen nature of the positive reactions obtained in the skin, failed in three cases. Successful results, although frequent in urticaria with this

method, have only rarely been obtained in cases of dermatitis. Biberstein has, however, actually demonstrated the Prausnitz-Küstner reaction in ursal dermatitis. A negative Prausnitz-Küstner reaction merely suggests that demonstrable antibodies are not present in the serum, and does not exclude the probable antigen-antibody nature of the allergic reaction of the skin cells.

The facts observed with compound "A" therefore show that any skin reaction which occurs with this substance in low concentration is due to the allergic conditions of the skin, and not to the primary irritant action of "A." There are ample opportunities for "A" present in a fur to dissolve in the secretions of the skin when the fur is brought into contact with the skin, and it is probable that the allergic skin only reacts to "A" when in solution.

The latent period observed clinically between the first time of wearing a fur and the development of dermatitis, which may amount to a few weeks, may be due to several factors. When short, it is probably a delayed reaction to the first contact with the fur, similar to that noted in the reaction to skin tests. When of longer duration, it may either be due to an initially non-allergic skin developing allergy, as has been shown to be possible in the preliminary experiments, or to the fact that in an allergic person no direct contact between the skin and the dyed fur has occurred during the first two or three occasions on which the fur has been worn.

The results obtained in the foregoing experiments suggest that similar processes are responsible for the production of dermatitis by furs dyed with other amines, diamines, aminophenols, diphenols, and their substitutes. No definite comparisons are justifiable, however, and any fur which has been associated with dermatitis must be subjected to similar biological analysis before a definite statement can be made regarding the nature and cause of the dermatitis. Chemical analysis of the fur yields no information as to its possible biological effects. It is a well-known clinical fact, and one which is illustrated by the case-histories of the fur-sensitive cases here reported, that an individual may suffer from dermatitis when wearing one dyed fur, but not another. The reason for this may be that the pre-existing or subsequently developed allergic state of the patient's skin is specific for one amine body, or that the fur which is not associated with dermatitis contains no available antigen.

It has already been pointed out that the experiments with "A" show that this substance is not, strictly speaking, an irritant, and that when signs of inflammation follow its application to the skin in an idiosyncratic person this is entirely due to the skin being in a highly intolerant or allergic state. From the series of tests carried out with "A" dyed fur and with "A" on unselected cases of skin diseases, it appears that a very small proportion of individuals exhibit an idiosyncracy to "A," and when the number of cases of fur dermatitis is compared with the total number of fur-wearers this proportion is infinitely less. Incidentally, it has been shown that the existence of various types of skin disease, other than fur dermatitis, are not associated with an unduly high proportion of individuals sensitive to "A." When several substances to which individuals are known to have shown an idiosyncrasy are compared, it is found that the number of idiosyncratic individuals varies greatly according to the substance. Thus Bloch[7] quotes experiments to show that 80 per cent. of all individuals are sensitive to ascaris, 60 per cent. to poison ivy, and 45 per cent. to orthoform. Moreover, by using concentrated extracts of primula, he has been able to produce an allergic state in the skin of 100 per cent. of persons, thus overcoming the individual constitutional factor (idiosyncrasy), and converting an idiosyncrasy into a pure allergy. These facts would have to be born in mind when assessing the extent to which any compound in a dyed fur was capable of participating in or precipitating allergic reactions of the skin.

They also have an important medico-legal bearing. First, the fundamental rôle played by allergy in such cases, and, secondly, the proportion of normal to idiosyncratic individuals ought to be considered when compensation claims arise.

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MEDICINE AND THE LAW.

The Theft of Motor-cars.

In his report for 1930 the Commissioner of Police for the Metropolis has to confess that there was a 50 per cent. increase in the number of motor cars reported stolen. It is true that he can claim that recovery quickly follows in a very large percentage of cases; out of 4941 cars stolen during the year only 182 were not recovered. But this measure of success is not completely reassuring to professional men who must rely on the reasonable safety of their means of movement. No doubt, as the report says, the cars are often taken only for pleasure rides. Here it is satisfactory that these so-called "borrowings" are now a punishable offence by virtue of Section 28 of the Road Traffic Act. The Commissioner comments somewhat naïvely on the ease with which burglars and other criminals can facilitate their operations by simply appropriating a car left standing in the street. "Greater use," he says, "should be made of garages." But is a doctor expected to leave his car in a garage every time he visits a patient? If garaging is impracticable, adds the report, drivers leaving their cars unattended should take precautions to prevent them being driven away; "professional criminals dislike work and trouble, and it is almost unknown for a car to be stolen from the street when reasonable precautions have been taken." But the Commissioner is obliged to remind us that "it may be necessary in emergency for the car to be moved by hand in the owner's absence." Indeed, in the London traffic area car-owners have been prosecuted for locking their cars; they intended to frustrate the criminal but they broke statutory regulations. The original London Traffic (Parking Places) Regulations of 1925 merely said that a driver must move a parked car if required by a constable, and that "on the occasion of any public procession or for other good and sufficient reasons or in case of emergency" a constable might move a vehicle whether or not the driver was present. Fresh regulations in 1928 directed that, if a driver left a car unattended at a parking-place, he must so leave it that it may be "moved from one position to another by manual power"; it might be so moved by a police constable in uniform "or other person authorised by such constable" for any reasonable cause in the

absence of the driver. These provisions remain unaltered in the superseding regulations of 1929 now in force. It should be noticed that, in the driver's absence, the car is not to be moved otherwise than by hand; anything which makes it difficult for an unauthorised person to start the engine is therefore a legitimate precaution. It should also be noted that these regulations apply only to cars parked at the official parking-places in London scheduled to the 1929 regulations (as from time to time varied). There seems to be no law that a car left temporarily outside a patient's house must be so left that it can be moved even by manual power, though emergencies can be imagined in which such movement might be in the interests of the car-owner.

The use of the mobile squad and of wireless communication with patrols may make things harder for the car-thief. We must wait for next year's figures to see what improvement may be due to such measures. These criminal statistics for London show a serious position. Whether helped by stolen cars or not, burglaries rose from 262 in 1929 to 352 in 1930, and the breaking and entering of buildings other than houses rose from 1539 to 2191.

CORRESPONDENCE

THE CHEMOPROPHYLAXIS OF MALARIA.

To the Editor of THE LANCET.

SIR,—The gratitude of workers in tropical medicine all the world over is due to Colonel S. P. James and his co-workers, Dr. W. D. Nicol and Mr. P. G. Shute, for their convincing demonstration of the value of plasmoquine in therapeutic doses as a prophylactic against malaria infection. (THE LANCET, August 15th, p. 341). That plasmoquine* has a selective action on the sporozoites of malaria is an entirely new scientific fact and one of far-reaching importance. As one who has been actively engaged in endeavouring to assess the exact value of plasmoquine treatment in malaria subsequent to its introduction to therapeutics in 1926, this paper comes as a great encouragement.

There can be no doubt now that plasmoquine has a distinct effect upon the trophozoites of malaria in the blood, but even here, as far as my own observations go and as I have emphasised in my publications on the subject, the drug acts in a different manner to quinine. For instance, it appears to be more efficacious in extirpating the trophozoites of the benign tertian parasite than those of the quartan or subtertian. In fact in subtertian malaria the drug in full therapeutic doses of 0·06 to 0·08 g. daily appears to be unsatisfactory so that clinical and parasitic relapses have occurred in my experience whilst the patient is under treatment, as I reported in my original paper.[1] But the most remarkable effect of plasmoquine is its selective action upon the gametocytes of the malaria parasite upon which quinine (when given uncombined with plasmoquine) appears to act very slowly and with uncertainty. The crescent form, or gametocyte, of the subtertian parasite may be taken as an index of the particularly selective action of the drug which is at present unexplained. Crescents are known to persist for a long time, even for weeks, in the peripheral blood of patients when on full doses of quinine, as I reported in my second paper[2] they have been observed apparently unchanged after the ingestion of grs. 840 (56 g.) of quinine.

The peculiar action of plasmoquine on crescents was first noted by P. Mühlens[3] and later by myself it was figured in the paper just quoted. These observations have been strikingly confirmed by W. Mollow[4] who has given a coloured plate of the curious appearance assumed by these defunct gametocytes. It was thought quite naturally at that time that, in the effective destruction of gametocytes and the sterilisation of the blood resulting therefrom as a means of infecting anophelines, the chief value of plasmoquine chemoprophylaxis (to use the new term) lay.

The combination of plasmoquine with quinine sulphate as first advocated by W. Schulemann and G. Memmi[5] was a distinctive advance, especially in the treatment of subtertian malaria, for the combination with quinine enhanced the value of plasmoquine in the treatment of malaria and apparently counteracted the well-known toxic action of plasmoquine when given uncombined. I have strongly advocated the use of this combined treatment in the routine treatment of malaria in a third paper.[6] This view was then considered too optimistic, but has since received ample confirmation. It is probably true that results obtainable by "plasmoquine compound" treatment in a cool climate are not comparable with those recorded in the tropics, where the climatic and dietetic factors are a detriment to its vis medicatrix and the ultimate recovery of the patient; moreover, it is probably true too, that the initial doses of plasmoquine advocated, 0·08–0·1 g. daily, were far too large. I am now obtaining equally satisfactory results with much smaller doses, such as 0·04 g. plasmoquine daily combined with 0·5 g. quinine sulphate (grs. 7½). Some of the most gratifying results I have had have been in the treatment of malaria in pregnancy, especially in women infected with subtertian malaria where the danger to mother and fœtus is well known. The drug appears to be remarkably well tolerated, and in my experience, now of several cases, leads to the termination of a normal pregnancy and labour without relapses. I have had also satisfactory results in pregnancy with quartan and benign tertian infections and I hope soon to publish these results in extenso.

For the last three years I have advocated the use of plasmoquine compound as a prophylactic to patients returning to the tropics to malaria-infested stations, especially in Central and West Africa, and reports I have had recently from patients who have returned after a tour of service have been most satisfactory. Naturally, I have not had the opportunity of testing my observations upon a large and scientifically conducted scale. My advice has been to take one tablet of plasmoquine compound (plasmoquine 0·01 g.

* There has been some confusion about the name and composition of this drug. We are assured by Bayer Products, Ltd., the London agent of its makers, that Plasmoquine, as now issued, is identical with the preparation in the first instance issued under the name of Beprochin. There has been no alteration in its composition. Plasmochin is simply the continental spelling of Plasmoquine. The makers state that Plasmoquine is N-diethylamino-isopentyl-8-amino-6-methoxy-quinoline, obtained synthetically. It is not derived from quinine.—ED. L.

[1] Proc. Roy. Soc. Med., 1927, 919–926.
[2] THE LANCET, 1928, i., 25.
[3] Beiheft z. Arch. f. Schiffs- und Tropen-Hyg., 1926, xxx., 305.
[4] Arch. f. Schiffs- und Tropen-Hyg., 1928, xxxii., 116.
[5] Klin Woch., 1927, vi., 1093.
[6] THE LANCET, 1928, ii., 496.

plus quinine sulphate 0·125 g.) last thing at night on going to bed during the malaria season. It is specially important to observe that the drug should never be taken on an empty stomach.

Obviously these valuable observations of Colonel James are going to impart a new stimulus to this aspect of the subject and will probably result in the elaboration of even more satisfactory compounds of plasmoquine and in setting the true and scientific prophylaxis of malaria on a proper and practical basis.

I am, Sir, yours faithfully,

PHILIP MANSON-BAHR.

Weymouth-street, W.1, August 14th, 1931.

IMPRISONMENT FOR MENTAL DISORDER.

To the Editor of THE LANCET

SIR,—I am glad that Prof. Millais Culpin has drawn public attention to the unfortunate man who was sentenced at Hove Police-court to fourteen days' imprisonment for an offence committed when he was alleged to be suffering from loss of memory. On the day following the conviction I telephoned to the Home Office and urged that the case should be investigated. A member of the secretarial staff listened courteously to my plea and promised to look into the matter.

I cannot agree with Prof. Culpin, however, that no blame attaches to the police or the bench. Surely it is not asking too much of the magistrates to expect them to recognise that a man who genuinely fails to know his name and family is in an abnormal mental state, and that the genuineness of his amnesia and its bearing upon his criminal responsibility are matters which should be the subject of medical evidence.

I am, Sir, yours faithfully,

W. RUSSELL BRAIN.

Harley-street, W., August 17th, 1931.

THE LATE LIEUT.-COLONEL LORD.

To the Editor of THE LANCET.

SIR,—I desire the favour of a few lines in which to express the admiration I had, shared with many other members of the Royal Medico-Psychological Association, for the late Lieut.-Colonel Lord. I can speak of him as a friend and colleague in the Medico-Psychological Association and in the Council of Mental Hygiene.

An outstanding feature of his character that could not be overlooked was Colonel Lord's unbounded interest and enthusiasm in the work and welfare of the Association, which has been the means of placing our nurses and mental hospitals in the van of the civilised world. No time or trouble spent on behalf of the Association was grudged by him. A phrase of this kind is often used in complimentary addresses and appreciations, but in Colonel Lord's case it has a special meaning. He held an important post under the London County Council, and this involved a full day's work. Every evening, however, after his official duties for the day were over, he began a second day's work for the Association, and worked on till all the hours of the morning of the next day. The amount of work, mainly of a literary and organising kind, that he overtook during these undisturbed hours in the evening and the night-time, was colossal.

His official post in the Association for many years was that of editor of the *Journal of Mental Science*, itself affording enough scope for the energies of any ordinary man. He discharged the duties of this post—both from the business and scientific points of view—most efficiently, and enhanced the reputation of the journal. Some six years ago he was elected President of the Association and since then he has played an important, if not a leading, part in organising its schemes and enterprises. Nothing that was done by the Association was outwith his interest and activities. The preparation of evidence for the Royal Commission, the presentation of the views of the Association on the new Mental Treatment Act, the elaboration of a new constitution and laws for the Association, now designated Royal; these and similar operations were all influenced by his guidance. The training of mental nurses, their examinations, and their registration by the Association was one of his chief occupations, in view of the fact that their interests had been sacrificed by the General Nursing Council in favour of those of hospital nurses. A permanent memorial of his energy was the creation of the Research Committees of the Association, whereby excellent scientific work has been encouraged with success in our mental hospitals. Lastly, he was secretary and an active member of the Council of Mental Hygiene, and at the International Congress held at Washington last year, he did most useful work for Great Britain.

Colonel Lord did the work of two men, and that under very unfavourable physical conditions. His health during all these years was critical, and when he should have rested, he worked the harder. Being anxious on one occasion on account of his exhausted condition, I spoke to his niece, who then looked after his welfare, to order him to stop work. She answered sadly: "There is no living person who can get him to stop work." If ever a man died in harness, he did. Possibly he found distraction from his anxious physical condition in hard work. At all events, for years he was the most notable example known to me of a brave man struggling against adversity, refusing all compassion, and refusing to acknowledge defeat. His work over, he now rests.

I am, Sir, yours faithfully,

GEORGE M. ROBERTSON.

Edinburgh, August 16th, 1931.

EXCESSIVE SPONTANEOUS INFLATION OF A LUNG CAVITY.

To the Editor of THE LANCET.

SIR,—I read with interest Dr. Blyth Brooke's excellent account of a case diagnosed as excessive spontaneous inflation of a lung cavity (THE LANCET, August 1st, p. 240). One may note that this condition has been observed with manometric control by Dr. Vere Pearson [1] who regards it as a factor in the normal pathogenesis of pulmonary cavities. Dr. W. Mitchell [2] has also concluded that the cavities previously known as pleural rings, which he calls air cysts, are produced possibly by a check-valve action in the ulcerated wall of a bronchus or bronchiole. Such a cavity has been investigated by Dr. Andrew Morland, who informed the Tuberculosis Association last June that he had found a positive pressure in it.

Dr. Blyth Brooke remarks that the diagnosis in his case was between inflated cavity and spontaneous pneumothorax, and suggests that dullness and cavernous breathing posteriorly tend to exclude pneumothorax. However, in such a case as he describes, one would expect adhesions posteriorly to prevent a complete spontaneous pneumothorax, and

[1] Brit. Med. Jour., 1930, i., 380.
[2] Brit. Jour. Radiol., 1930, iii., 446.

these would also tend to anchor the mediastinum. Consequently, I should suppose that, judging from his account, Dr. Blyth Brooke inserted his needle either into a compressed cavity or, more probably, through lung into an anterior pneumothorax.

One would be disposed to criticise Dr. Blyth Brooke, first, in his choice of the posterior route through lung into the cavity, for there would have been no risk in using an initial needle anteriorly; secondly, in his advocacy of the introduction of pneumothorax needles through lung into dilated cavities for the relief of pain. Such a method would normally be a temporary expedient, for the check-valve mechanism would remain, and also it would occasionally cause unpleasant hæmoptysis.

Dr. Blyth Brooke suggests that "in view of the extent of toxæmia, it was considered that neither an artificial pneumothorax nor a phrenic evulsion would offer her any material assistance." Does he then regard it as a general principle that a patient with a unilateral lesion, who is in a very toxic state, is unsuitable for pneumothorax? Surely very careful collapse therapy is exactly what is indicated.

I am, Sir, yours faithfully,

H. W. ALLEN.

Bryanston-street, W., August 11th, 1931.

OSTEO-ARTHRITIS.

To the Editor of THE LANCET.

SIR,—In Dr. Philip Figdor's paper on p. 382 of your last issue, he makes the statement that this disease "attacks people who have reached the age of double dentures." I think one may infer from this that Dr. Figdor considers that such people cannot have any oral sepsis. With this point of view I cannot agree, for septic foci in completely edentulous alveolar margins are by no means uncommon, and are as much (if not more) a source of septic infection as abscessed teeth. I called attention to this in a short note in your issue of May 17th, 1930. Dr. Figdor goes on to say that "in those who have teeth, extraction of the remaining teeth seems to make very little difference." Unless these teeth were infected, I do not suppose that it would make any difference. I cannot think that trauma alone is sufficient to account for all the changes found in osteo-arthritis. Would it not be more reasonable to suppose that the trauma prepared a way for microbic action?

I am, Sir, yours faithfully,

J. D. KIDD, M.B.

Queen Anne-street, W., August 14th, 1931.

ANÆSTHESIA IN LABOUR.

To the Editor of THE LANCET.

SIR,—With reference to the communication by Dr. Jane King and Dr. Joyce Morgan on page 346 of your last issue, I have found that satisfactory results can be obtained in private practice without the use of an expensive apparatus. All that is necessary to carry is a 100-gallon cylinder of nitrous oxide. For a small cost a reducing valve can be provided for the cylinder, automatic filling valves for the bag, and a self-administering stop-cock between the bag and the facepiece. Administration can be started towards the end of the first stage. When a uterine contraction begins four deep breaths are taken from the bag, the breath being held after the last to prolong the effect of the gas. The facepiece is then laid aside until the next contraction begins. By this method analgesia is produced. Cyanosis is slight and passes with the next breath of air. An oxygen cylinder is not required.

Labour is not prolonged. Full anæsthesia for the birth of the head is procured with chloroform. In normal cases the help of an anæsthetist is not essential, the final anæsthesia being entrusted to the nurse. In abnormal cases I have the assistance of an anæsthetist. I am, Sir, yours faithfully,

JAMES RIDDELL, M.D., F.R.C.S. Edin., M.C.O.G.

Plymouth, August 16th, 1931.

LORD KNUTSFORD REMEMBRANCE FUND.

To the Editor of THE LANCET.

SIR,—A letter has been received at the London Hospital enclosing a donation of £1000 and urging that a fund should be started in remembrance of Lord Knutsford. This is but one instance of a very general desire. Many other gifts for the same purpose have already been received, as have a number of letters asking for an opportunity to contribute. We know, too, that collections by groups of workers are being made spontaneously. The committee have therefore decided to open such a fund. There were, at the moment of his death, certain very necessary additions and improvements on which Lord Knutsford had set his heart and to which he had contributed generously, but which he did not live to see completed. It is proposed to devote gifts sent in remembrance to the completion of these objectives, which include a new massage and electrical department, new stores, additional maternity beds and maintenance, and a hostel for students. Will all who wish to honour the name and work of a great man send their contributions to me at the London Hospital, E.1?

I am, Sir, yours faithfully,

EDWARD J. MANN,

August 17th, 1931. Treasurer.

BENDIEN'S WORK ON CANCER.

To the Editor of THE LANCET.

SIR,—During a visit to Zeist, Dr. Bendien has given me ample opportunity of discussing his discovery with him, of seeing him perform his tests, and of talking to and examining his patients. He does not claim to have discovered a cure for cancer, but he believes, and he is most emphatic on this point, that his method of *diagnosis* of cancer in the early and later stages are infallible. He believes carcinoma to be a local disease which can develop only if a specific alteration or, as he puts it, "specific stabilisation" of the serum is present. This abnormality of the serum he can detect by his diagnostic methods. The treatment therefore consists in restoring the blood to its normal condition, thus producing a medium unsuitable for the growth of cancer. He advises operation in operable cases; his treatment after operation, as shown by his serological tests prevents recurrences.

The diagnostic test consists of two parts—namely, flocculation of the serum, and spectro-photometric examination of the precipitate. The first part, the flocculation, which is described in your issue of May 16th, p. 1097, Dr. Bendien regards rather as a preliminary, although in experienced hands it may be sufficient for a diagnosis of cancer. He tells me that in the series of 20 test-tubes containing standard mixtures of acetic acid and sodium vanadate (1) normal human blood-serum always flocculates, if

not heated, at No. 6; (2) a serum which flocculates below No. 6 indicates a pathological condition such as carcinoma or tuberculosis; and (3) the lower the number at which flocculation takes place the more concentrated is the "toxic agent" of the serum. Cases flocculating below No. 4 are of particularly bad omen; these are the cases which, according to Dr. Bendien, are inoperable, not in a technical sense, but because dissemination has already taken place, as shown by the great quantity of "toxic" material present in the serum.

The spectro-photometric test, employed for accuracy of diagnosis and as a confirmation of flocculation, is a highly technical process involving mathematical calculation. The precipitate obtained with the acetic vanadate mixture is heated to 56° C. to dissolve what Dr. Bendien calls "normal-labilin" (a protein present in all sera); it is filtered off, weighed, and suspended in $NaHCO_3$ (1 : 50). Suspensions, 20 in all, of various strengths are now submitted to spectro-photometric examination, and 20 photographs of the ultra-violet part of the spectrum are taken. The absorption lines are plotted on a chart and a curve so obtained which is typical of cancer, indicating the presence of a thermostable protein peculiar to it.

Dr. Bendien kindly gave me a full account of the methods and principles of his treatment, but it is impossible for me to go into details here. He wishes me to state that all cases so far treated were inoperable ones, generally with metastases; most of these cases had been submitted to radium, X rays, &c., without success before they came under his care. But in every case he was able with his treatment to obtain an improvement: he does not wish to make any other claim.

I am, Sir, yours faithfully,

A. A. MILLER, M.D., M.R.C.S.

Harley-street, W., August 17th, 1931.

THE SERVICES

ROYAL NAVAL MEDICAL SERVICE.

Surg. Lt. F. C. Crean to be Surg. Lt.-Comdr.

ROYAL NAVAL VOLUNTEER RESERVE.

Surg. Lts. B. W. C. Archer and H. Winstanley to be Surg. Lt.-Comdrs.

Surg. Sub-Lt. P. B. Moroney and Proby. Surg. Lt. W. R. Johnston to be Surg. Lts.

ROYAL ARMY MEDICAL CORPS.

Lt. W. A. R. Ross to be Capt. and remain secd.

The undermentioned Lts. (on prob.) are confirmed in their rank: D. A. Langhorne, C. A. de Candole, and W. B. Williams.

ARMY DENTAL CORPS.

The undermentioned Lts. (on prob.) are confirmed in their rank: S. G. Watson and G. T. Drummond.

ARMY RESERVE OF OFFICERS.

Col. A. Chopping, late R.A.M.C., having attained the age limit of liability to recall, ceases to belong to the Res. of Off.

TERRITORIAL ARMY.

Col. J. G. Martin, having attained the age limit, retires and retains his rank, with permission to wear the prescribed uniform.

Capt. G. W. Wigg from 72nd (Northumbrian) Fd. Bde., R.A., and Capt. K. J. T. Wilson (late R.A.M.C. Spec. Res.), to be Capts.

ROYAL AIR FORCE.

The undermentioned are granted permanent commissions in the ranks stated: Flight Lt. J. H. Cullinan and Flying Officers C. G. Harold and A. Sheehan.

ROYAL AIR FORCE RESERVE.

Flying Officer C. J. MacQuillan is promoted to the rank of Flight Lt.

INDIAN MEDICAL SERVICE.

Lts. to be Capts.: L. F. Burns, L. Dass, P. C. Dutta, and E. A. O'Connor.

Lt. (T.C.) A. M. H. Shirazi to be Capt. (T.C.).

The undermentioned appts. are made: to be Lts. (on prob.): E. H. Lossing, G. W. Miller, D. G. McCaully, and H. J. R. Thorne.

Lt.-Col. C. J. Stocker retires.

COLONIAL APPOINTMENTS.

The following appointments have been made by the Secretary of State for the Colonies: Dr. R. H. Davidson, M.O., and Dr. T. A. P. Wynter, Government M.O., Jamaica; and Dr. C. A. Stanley, M.O., Malay States; Dr. B. N. V. Bailey, Asst. Government M.O.H., British Guiana, has been promoted Government M.O.H.; Dr. S. G. Harrison, M.O., Gambia, has been transferred to the Gold Coast; and Dr. J. T. Smalley, M.O., Hong-Kong, has been promoted senior M.O.

INFECTIOUS DISEASE

IN ENGLAND AND WALES DURING THE WEEK ENDED AUGUST 8TH, 1931.

Notifications.—The following cases of infectious disease were notified during the week:—Small-pox, 25 (last week 26); scarlet fever, 1250; diphtheria, 759; enteric fever, 62; pneumonia, 472; puerperal fever, 41; puerperal pyrexia, 97; cerebro-spinal fever, 25; acute poliomyelitis, 8; encephalitis lethargica, 11; dysentery, 6; ophthalmia neonatorum, 109. No case of cholera, plague, or typhus fever was notified during the week.

The number of cases in the Infectious Hospitals of the London County Council on August 11th–12th was as follows: Small-pox, 46 under treatment, 2 under observation (last week 54 and 2 respectively); scarlet fever, 1159; diphtheria, 1455; enteric fever, 14; measles, 145; whooping-cough, 364; puerperal fever, 42 (plus 8 babies); encephalitis lethargica, 211; poliomyelitis, 3; "other diseases," 135. At St. Margaret's Hospital there were 11 babies (plus 5 mothers) with ophthalmia neonatorum.

Deaths.—In the aggregate of great towns, including London, there was no death from small-pox, 1 (1) from enteric fever, 12 (1) from measles, 1 (0) from scarlet fever, 15 (6) from whooping-cough, 25 (4) from diphtheria, 33 (7) from diarrhœa and enteritis under two years, and 16 (1) from influenza. The figures in parentheses are those for London itself.

Two deaths from measles were reported from Barnsley, not more than 1 from any other large town. Liverpool reported 5 deaths from whooping-cough and 4 from diphtheria. Diarrhœa was credited with 6 deaths at Birmingham, 3 at Manchester, and 2 at West Hartlepool.

The number of stillbirths notified during the week was 266 (corresponding to a rate of 46 per 1000 births) including 58 in London.

MIDDLESEX POOR-LAW HOSPITALS.—Each of these institutions has had the word "County" added to its name, with the exception of Park Royal Hospital, which will henceforth be called "The Central Middlesex County Hospital." The domestic accommodation at the West Middlesex County Hospital is to be improved at a cost of nearly £13,000.

NATIONAL BIRTH CONTROL ASSOCIATION.—The National Birth Control Council and the Birth Control Investigation Committee have amalgamated under the title of the National Birth Control Association (with which is incorporated the Birth Control Investigation Committee). Sir Thomas Horder has accepted the presidency of the Association, and Sir Humphry Rolleston, who remains chairman of the Birth Control Investigation Committee, will be vice-chairman. The committee has been for over four years investigating the general problem of birth control, as well as the merits and demerits of different methods, while the Council, which was founded last year, has been engaged mainly on work with local authorities in regard to the Ministry of Health's memorandum on birth control. It is stated that so far 35 local authorities have authorised the giving of advice on contraceptive methods. The office is at 26, Eccleston-street, London, S.W. 1.

OBITUARY

WALTER ERNEST DIXON, O.B.E., M.D., F.R.C.P. Lond., F.R.S.

Dr. W. E. Dixon died last Sunday at his home in Cambridge, and the loss will be widely felt. He long played a notable part in the work of the Cambridge medical school, and as expert adviser his exceptional knowledge and experience were freely available in the service of medicine, both national and international.

DR. W. E. DIXON.

[*Photograph by J. Palmer Clarke.*

The youngest son of Mr. Robert Bland Dixon, of Darlington, Walter Ernest Dixon was educated at Darlington, Dulwich, and St. Thomas's Hospital. He graduated as B.Sc. Lond. in 1891, and as M.B. five years later. After house appointments at St. Thomas's he held posts in the physiological department, and soon began to turn his mind more and more to the action of drugs. At this time pharmacology was receiving little attention or encouragement in this country, but he was granted a Salters Research Fellowship, and began to publish experiments. In due course he became professor of materia medica and pharmacology at King's College, but only temporarily, for he was more attracted by the opportunities for investigation offered by Cambridge. He had taken the D.P.H. there in 1898, and in 1902 he was appointed assistant to the Downing professor of medicine, receiving the honorary degree of M.A. In 1909 he became lecturer, and in 1919 reader in pharmacology, and as teacher, writer, and research worker he came to occupy a distinguished place among the pharmacologists of the world.

As a teacher he exerted a profound influence over generations of students, especially in the way of inculcating the physiological basis of therapeutics. At the outset of his Cambridge courses he captured the attention of undergraduates, and he retained it to the very end. Pharmacology became the most fascinating of studies, because Dixon, with his cynical humour, was one of the best of lecturers. So stimulating an instructor was much in demand outside his own university, while at the same time nearly every medical school in the country invited him to become an examiner. In this capacity he was often regarded with alarm, and his methods did nothing to encourage the candidates' self-conceit, but he was always fair, and even lenient in his final decision.

Dixon was a prolific writer, and contributed to Hale White's "Text-book of Materia Medica," Heffter's "Pharmacology," Allbutt's "System of Medicine," and the British Pharmaceutical Codex, to mention only a few. His "Manual of Pharmacology," which has passed through many editions, has been the avenue through which medical practitioners all over the country have approached their examination in this subject. He succeeded Cushny as the senior editor of the *Journal of Pharmacology*, and was on the editorial committee of several other scientific journals. He also served on many important commissions and committees, including that which the League of Nations set up to consider drug addiction. In this capacity he was valued not only for his authoritative knowledge but also for his faculty of singling out the essential points of a problem.

As an investigator, he made many contributions to physiology and pharmacology which covered a wide field. Among them may be mentioned his researches on apocodeine, tyramine, histamine, and digitalis, on general problems of pharmacology, especially tolerance and site of action of drugs; but of chief importance was his pioneer work on the physiology and pharmacology of the bronchial muscle and on the cerebro-spinal fluid. In all his researches he brought to bear a fearless and original mind, never afraid to question tenets apparently established, and he devised many improvements in pharmacological technique. He had, moreover, the faculty of gathering young men around him and interesting them in research work. He was full of ideas, often heterodox, and many of his old pupils, who now occupy important teaching positions, would willingly acknowledge his help, guidance, and inspiration.

Dr. O. Inchley, lecturer on pharmacology at King's College, London, writes: "On the title page of his 'Manual of Pharmacology' appears a quotation from Renan, 'Dire n'est rien; faire est tout,' which, to me, gives the keynote of Dixon's life-work. Not long ago the assertion of physicians as to the beneficial action of this or that drug in this or that disease was taken on faith. To-day the actions of drugs are determined physiologically by experimental methods, and in the birth of this modern science of pharmacology Dixon took an outstanding position. His manual contains liberal illustrations of kymograph tracings, each in itself an accurate experimental record and supplying the student with facts rather than opinions. The simple experiments suitable for class work set out in his 'Practical Pharmacology' convince the student, by his own observation, of the action of the drug on the animal. Dixon's lectures were never stodgy. His personality insured that whatever he touched was raised from the commonplace. We, who knew him intimately, knew that whenever he was with us we should never be bored. Dixon at the dinner table, Dixon on a walking tour, or Dixon on a rock scramble in Sark was the leader, life, and soul of the party. His wit was sprightly and never cruel. He excelled as a raconteur, and some of his most amusing tales were against himself."

"The subject of pharmacology," writes Prof. J. A. Gunn, "has suffered grievous losses in the last few years—Cushny, Magnus, Loevenhart, Trendelenburg, and now Dixon, all men in the front rank, and all, one hoped, with fruitful years of work still before them. For one who has enjoyed an uninterrupted friendship with Dixon for over a quarter of a century, and who has experienced nothing but kindness and encouragement from him, it is difficult at present to write any coherent appreciation of his work. He had come to be accepted, since Cushny's death, as the leader of academic pharmacology in this country, and his loss, when we have so few pharmacologists,

and indeed few medical men with his wide training and outlook, is in the meantime irreparable. No one who knew him will forget his arresting personality. A wide and unusual knowledge of men and things, a gift for telling stories, an infectious and almost boyish enthusiasm, made him a delightful and resourceful companion."

Dr. Dixon became an F.R.S. in 1911. In 1930 he was elected F.R.C.P., and he was recently appointed to deliver the Croonian lectures. At one time and another he was president of the section of pharmacology of the British Medical Association, president of the section of therapeutics of the Royal Society of Medicine, and president of the section of physiology of the British Association. Last year he received the honorary degree of LL.D. of Manitoba. During the war, with a nominal appointment in the Royal in the Navy, he played an obscure but important part intelligence department, especially in connexion with secret service in Spain. For this he was created O.B.E.

At the time of his death Dr. Dixon was 60 years of age. He married in 1907 the only daughter of the late Mr. Francis Glen-Allan, of Dulwich, and their house at Whittlesford was known for its generous hospitality.

MARTIN WILLIAM FLACK, C.B.E., M.B. Oxf.,

DIRECTOR, MEDICAL RESEARCH DEPARTMENT, R.A.F.

WE regret to record the death on Sunday last of Group-Captain Martin Flack, the distinguished physiologist, who became the first director of the Medical Research Department of the Royal Air Force.

Martin William Flack, the son of Mr. E. C. Flack, of Borden, Kent, was born in 1882, and received his early education at the Great Yarmouth and Maidstone Grammar Schools. He proceeded to Keble College, Oxford, with a science exhibition, and took up the study of medicine, coming under the influence of such potent teachers as the late Prof. Gotch, Prof. Arthur Thomson, and Prof. J. S. Haldane. He graduated in Arts at Oxford in 1905, and completed his medical education at the London Hospital, where he held a scholarship. While at the London Hospital he was associated with Sir Arthur Keith in an investigation into the nerve-muscle structure of the heart,[1] and he also carried out, in collaboration with Sir Leonard Hill, a preliminary inquiry into the physiology of respiration. He graduated in medicine at Oxford in 1909, and was elected to a Radcliffe Travelling Fellowship. In accordance with the terms of the Fellowship he spent a year on the continent, mainly continuing his study of respiratory physiology, and on his return to England became assistant to Sir Leonard Hill, who was holding the chair of physiology in London University at the London Hospital Medical College. Flack was appointed lecturer in physiology and chemical pathology at the college, and later was associated with Hill's work at the Medical Research Council in the department of applied physiology. It was with these credentials that he came to the notice of the Air Ministry, who in the early days of the war were instituting a department of medical research specially directed to the elucidation of the problems of aviation as they affected the personnel of the Service.

GROUP-CAPTAIN FLACK.
[Photograph by Russell and Sons.

From the beginning Flack proved an unqualified success in a new and difficult appointment. He was saturated with the kind of knowledge whose application was most desirable, and had that knowledge wonderfully well sorted, for he had been a successful teacher of physiology, and was actually in the middle of writing a text-book with Sir Leonard Hill on the subject when the war broke out. He had assisted in valuable demonstrations of practical pathology, much of which bore directly upon the very questions which the new department would have to answer. His personal researches into important phenomena of the physiology of heart and respiration were exactly of the sort to stand him in good stead in devising tests which would assist in the recruiting of the candidates for the service. The report for 1920 of the Royal Air Force may be called in evidence for these statements. It contained a section describing a Medical Board for aviation candidates which showed how thoroughly Flack and his willing collaborators had faced the devising of a course of training and a method of inspection and examination which should result in securing the right men for a dangerous service—in essence the procedure was directed to removing, as far as possible, the element of danger. The result of this work was to make it necessary for the candidate to pass a medical examination to prove physical fitness, to provide evidence of good personal history, and to emerge successfully from a physiological assessment, based upon tests of the cardiac, respiratory, and nervous systems. An anthropometric index of physical fitness was devised, and standards were laid down which, though not unduly high, would lead to the inevitable rejection of recruits whose acceptance would be cruelty to themselves and dangerous to their colleagues. The first Director of Medical Research in the Royal Air Force Medical Service died while still holding the appointment which he was so adapted to fill and develop. He had borne a long illness in the Air Force Hospital at Halton before the end came in his fiftieth year. The Air Force owes him an inextinguishable debt.

Group-Captain Flack married Cecile, daughter of Mr. J. C. Cooper, and his widow survives him, with two sons and two daughters.

[1] THE LANCET, 1906, ii., 359.

SHEPHERD McCORMICK BOYD, F.R.C.S. Irel.

THE death of Dr. Shepherd Boyd, of Harrogate, on August 8th, will be regretted by many whom he befriended and many who appreciated his unusual charm. Seventy-five years of age, he was the son of Dr. John William Boyd, once the oldest doctor in Ireland, who died at the bedside of a patient after 57 years of practice from one house. After attending Kilkenny College and Devon County School the son went to Trinity College, and distinguished himself as one of the best oarsmen Dublin has produced. Twice he reached the final of the Diamond Sculls at Henley, and in other branches of athletics his achievements

were sound, if not so brilliant. He qualified in 1880, became F.R.C.S. Irel. three years later, and saw many different sides of life as practitioner in his home town of New Ross, medical officer of Cork-street Fever Hospital during a small-pox epidemic, ship's surgeon on the Nile, resident at Manchester Maternity Hospital and Chelsea Dispensary, and surgeon to a mining company in Yorkshire and an ironworks in Wales. He practised in Manchester for some years, moved to Bexhill, and finally settled at Harrogate some 27 years ago. But his life as a spa practitioner was yet to be interrupted by the war. From youth onwards an enthusiastic volunteer and territorial, he was well "over age" on the outbreak of war, but nevertheless got to France and remained there until after the Armistice. During an air-raid in Boulogne his hearing was badly damaged, but in spite of this disability he joined an ambulance train in 1918 and survived an almost direct hit by a bomb at Étaples. Deafness, largely, if not entirely due to this war service, was a serious trial during the latter part of his life, and he had to contend with other illness. "But," writes a colleague, "he presented a picture of triumphant personality dominating by its own inherent strength the lesser tragedies that might well have daunted a lesser man. . . . He was marked out by an extraordinary kindness of nature, and no trouble was too great for him to take to help anyone who appealed for his sympathy or his aid. Coupled with this kindly nature was a sparkling and irrepressible gaiety of spirits." He was especially encouraging and helpful in his treatment of recently qualified members of the profession.

Dr. Shepherd Boyd was long associated with the Irish Medical Schools and Graduates Association, and the Association of British Postal Medical Officers, and was at one time president of the Harrogate Medical Society. His wife survives him, and a son.

Dr. WILLIAM BURNS, who died on August 4th whilst attending a patient in the dispensary at Glengall-street, Belfast, was a student of Queen's College, qualifying at Glasgow in 1901. He then volunteered for special plague duty in India, returning after two years to take up general practice in Belfast. In 1910 he took the D.P.H. Belf., with a view to appointment as part-time medical officer of health for the urban district. Dr. Burns is described by a colleague as one whose joy in life endeared him to his colleagues, while his devotion and skill produced a host of patients who regarded him not only as their medical adviser, but as a friend to whom they could bring all their difficulties. Thousands of persons lined the route when his body was laid to rest amid the beautiful surroundings of Knockbreda. Dr. Burns was in his sixty-first year.

MEDICAL PRACTICE IN CHINA.—It is announced that foreign medical practitioners wishing to practise in China should forward their examination diploma and any other documents attesting their qualifications to the nearest consul of their nationality for his inspection, with a request that he issue a certificate. When this certificate has been obtained, it should be forwarded, together with a statement of the applicant's record, two 2-in. half-figure photographs, a registration fee of five dollars, and a stamp-tax fee of two dollars, to the appropriate local government department (i.e., the health bureau, or, if the district has no health bureau, then the Public Safety Bureau or the district government) for transmission to the health section of the Ministry of the Interior.

MEDICAL NEWS

THE annual general meeting of the Medical Sickness Annuity and Life Assurance Society Ltd., will be held at the First Avenue Hotel, High Holborn, London, W.C., on Wednesday, Oct. 7th, at 4.30 P.M.

London Hospital Saturday Fund.

The annual street collections for this fund will be held on Sept. 26th.

University of Manchester.

The Prof. Tom Jones Memorial Fellowship in Surgery has been awarded to Dr. A. L. Kenyon.

St. Mary's Hospital Dinner.

The annual dinner of past and present students will be held at the Trocadero Restaurant, London, W., on Friday Oct. 2nd, at 7 for 7.30 P.M. Sir William Willcox will preside.

Royal Sanitary Institute.

Lord Leconfield, Lord Lieutenant of Sussex, and chairman of the West Sussex County Council, has consented to act as president of the forty-third congress of the Institute, to be held at Brighton from July 9th to 16th, 1932.

Post-graduate Courses in Berlin.

From Oct. 5th onwards a number of international post-graduate courses will be held in Berlin on general and special subjects. The latter include pædiatrics, urology, medical psychology, intrathoracic surgery, and physical and dietetic treatment. In the spring there will be further courses on radiology, surgery, obstetrics, industrial medicine, and ophthalmology. Particulars may be had from the information bureau of the Kaiserin Friedrich-Haus, Luisenplatz 2, Berlin, N.W. 6.

Manchester Radium Institute.

Dr. Ralston Paterson has been appointed director of the Manchester Radium Institute in succession to the late Dr. A. E. Birkett. He will also be director of the national radium centre at Manchester, and will superintend the use of radium lent by the Radium Trust to the Royal Infirmary and the Institute. Dr. Paterson, who is at present working at the Edinburgh Royal Infirmary, has lately spent four years in the United States and Canada, and has made an extensive study of radiological methods there employed.

Congress of Tropical Medicine.

The second International Congress of Tropical Medicine will be held at Amsterdam from Sept. 12th to 17th, 1932, under the presidency of Prof. G. Grijns. The subjects of discussion will be (1) avitaminoses, with special reference to beri-beri; (2) yellow fever and leptospiroses; (3) helminths, with special reference to ancylostoma; and (4) malaria, with special reference to blackwater fever. Those wishing to participate in the Congress are asked to inform the general secretary (Prof. E. P. Snijders, Institute of Tropical Hygiene, Mauritskade, 57, Amsterdam) before the end of this year.

Fellowship of Medicine and Post-Graduate Medical Association.

The following are the subjects of special courses to be held in September: diseases of the chest at the Brompton Hospital, from Sept. 7th to 12th (all day); psychological medicine, at the Bethlem Royal Hospital, from Sept. 8th to Oct. 3rd (Tuesdays and Saturdays at 11 A.M.); diseases of infants, at the Infants' Hospital, from Sept. 14th to 26th (every afternoon); ophthalmology, at the Central London Ophthalmic Hospital, from Sept. 8th to Oct. 3rd (every afternoon); and medicine, surgery, and the special departments, at the Westminster Hospital, from Sept. 14th to 26th (all day, men only). Syllabuses of these courses may be had from the secretary of the Fellowship at 1, Wimpole-street, London, W.1. A series of free lectures on Prognosis will begin early in October, and also the usual series of free demonstrations in medicine and surgery—one a week in each subject at different hospitals.

THE LANCET: SUBSCRIPTION RATES.

INLAND	One Year	£2 2 0
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	Three Months	0 12 6

Medical Diary

Information to be included in this column should reach us in proper form on Tuesday, and cannot appear if it reaches us later than the first post on Wednesday morning.

LECTURES, ADDRESSES, DEMONSTRATIONS, &c.

FELLOWSHIP OF MEDICINE AND POST-GRADUATE ASSOCIATION, 1, Wimpole-street, W.

MONDAY, August 24th, to SATURDAY, August 29th.—ALL SAINTS' HOSPITAL, Vauxhall Bridge-road, S.W. Special post-graduate course in Urology. Afternoons and some evenings. Demonstrations and cystoscopies. —QUEEN'S HOSPITAL FOR CHILDREN, Hackney-road, E. All day course in Diseases of Children, Medical and Surgical demonstrations.—Further particulars from the Fellowship of Medicine.

ST. MARK'S HOSPITAL, City-road, E.C.

THURSDAY, August 27th.—4.30 P.M., Dr. Norman Henderson: The Lipiodol Investigation of Fistulæ.

Appointments

STANDRING, HELEN, M.D., Ch.B. Liverp., D.P.H., has been appointed Senior Medical Officer in charge of the Maternity and Child Welfare Department, Hull.

SUTHERLAND, R., M.B., Ch.B. Aberd., D.P.H., Asst. M.O.H., Aberdeen.

WILLIAMSON, A. B., M.D., Ch.B. Edin., D.P.H., Deputy M.O.H., Portsmouth.

Certifying Surgeons under the Factory and Workshop Acts:—BRADLEY, G. F., M.B., B.S. Lond. (Lutterworth); CLARKSON, H. G. H., L.R.C.P. Edin., L.R.C.S. Edin. (Ripley); GRIFFITHS, G. J. (Bedford); JONES, A. O., L.R.C.P. Lond., M.R.C.S. (Holywell); REID, A. C., M.B., Ch.B. Aberd. (Egremont); and SIMPSON, B. S., M.B., Ch.B. Edin. (Golspie, Sutherland).

Vacancies

For further information refer to the Advertisement columns.

Accrington, Victoria Hospital.—H.S. £150.
Albert Dock Hospital, Connaught-road, E.—Res. M.O. At rate of £110.
Altrincham, General Hospital.—Jun. H.S. At rate of £120.
Barnstaple, North Devon Infirmary.—Res. M.O. At rate of £150.
Bedford County Hospital.—Asst. H.S. At rate of £130.
Bethnal Green Metropolitan Borough.—Asst. M.O. £600.
Birmingham and Midland Eye Hospital, Church-street.—H.S. £110.
Birmingham, Canwell Hall Babies' Hospital.—Second Res. M.O. At rate of £200.
Bootle General Hospital, near Liverpool.—Cas. O. and two H.S.'s. All at rate of £150.
Bradford, Children's Hospital.—H.P. £120.
Bristol Eye Hospital.—Anæsthetist. Also Res. H.S. £150.
Bristol Royal Infirmary.—H.P.
Carnarvonshire C. C.—Deputy M.O.H. and Asst. School M.O. £700.
Cambridge, Addenbrooke Hospital. — Res. Anæsthetist and Emergency. At rate of £130.
Cardiff, King Edward VII. Welsh National Memorial Assn.—4 Asst. Tuber. O.'s. Each £250.
Carmarthen, Joint Counties Mental Hospital.—Jun. Asst. M.O. £350.
Coventry and Warwickshire Hospital.—Two Res. H.S.'s. Each £125.
Derby, Derbyshire Royal Infirmary.—Ophth. H.S. £150.
Dreadnought Hospital, Greenwich.—H.P. and H.S. Each at rate of £110.
Dudley, Guest Hospital.—Asst. H.S. £170.
Durham C. C.—Asst. Welfare M.O. £500.
East Ham Memorial Hospital, Shrewsbury-road, E.—H.P. At rate of £150. Also H.S. to Special Depts. and Cas. O. Each at rate of £120.
Edinburgh Royal College of Physicians.—Kirk Duncanson Fellowship for Medical Research. £150.
Edinburgh Royal Hospital for Mental and Nervous Disorders.—Asst. P. £350.
Edinburgh Royal Infirmary.—Clin. Assts. for Ear, Nose and Throat Department.
Golden Square, Throat, Nose and Ear Hospital, W.—Hon. Asst. Surg.
Great Yarmouth General Hospital.—H.S. £140.
Halifax, St. Luke's Hospital.—H.S. At rate of £250.
Hampshire County Council.—Asst. County M.O. £600.
Hastings, Royal East Surrey Hospital.—Jun. Asst. in Pathological Dept. £350.
Hayward Heath, King Edward VII. Memorial Eliot Hospital.—Surgeon to Ear, Nose and Throat Dept.
Hospital for Tropical Diseases, Endsleigh-gardens, W.C.—H.P. At rate of £150.
Hove, Lady Chichester Hospital.—Jun. H.P. At rate of £50.
Hull Royal Infirmary.—Third H.S. for Parent Institution. £150. Also H.P. Sutton Branch Hospital. £160.
Infants Hospital, Vincent-square, Westminster.—H.P. At rate of £75. Also Clin. Asst. for Out-patient Depts.
Leicester Royal Infirmary.—H.P. and H.S.'s. Also Cas. H.S. All £125.
Liverpool Eye and Ear Infirmary, Myrtle-street.—H.S. to Ophth. Dept. £120.
Liverpool Open Air Hospital for Children, Leasowe.—Jun. Res. M.O. £200.
London Temperance Hospital, Hampstead-road, N.W.—H.P. At rate of £100.
Longton Hospital, Staffs.—H.S. £150.
Maidstone, Kent County Ophthalmic and Aural Hospital.—H.S. £200.
Manchester, Hulme Dispensary, Dale-street.—Hon. M.O.
Manchester Royal Children's Hospital, Pendlebury.—Two Asst. M.O.'s for Out-patients' Dept. Each at rate of £150. Also Res. M.O. At rate of £125.
Manchester Royal Eye Hospital.—Jun. H.S. £120.
Margate and District General Hospital.—Res. M.O. £150.
Margate Royal Sea Bathing Hospital for Surgical Tuberculosis.—H.S. At rate of £200.
National Hospital for Diseases of the Heart, Westmoreland-street, Marylebone, N.W.—Res. M.O. Also Out-patient M.O. At rate of £150 and £125 respectively.
Newcastle-upon-Tyne, Hospital for Sick Children.—H.P. and Sen. and Jun. H.S.'s. All at rate of £100.
Newport, Mon., Cefn Mably Tuberculosis Hospital, Michaelstone-y-vedw.—Asst. Res. M.O. £250.
Norwich, Norfolk and Norwich Hospital.—H.S. and H.P. Each at the rate of £120.
Nottingham General Dispensary, Broad-street.—Res. Surg. £250.
Plymouth, South Devon and East Cornwall Hospital.—Res. Anæsthetist and H.S. At rate of £100.
Portsmouth, Royal Hospital.—Sen. H.S. £175. H.P. and Two H.S.'s. Each £130. Also Cas. O. £100.
Queen Charlotte's Maternity Hospital, Marylebone-road, N.W.—Dist. Res. M.O. At rate of £80.
Queen's Hospital for Children, Hackney-road, E.—Cas. O. At rate of £100. Also Asst. Surgeon.
Richmond Royal Hospital, Surrey.—Jun. H.S. At rate of £100.
Rotherham Hospital.—Cas. H.S. £150.
St. Leonards-on-Sea, Buchanan Hospital.—Jun. H.S. £125.
Scunthorpe and District War Memorial Hospital.—1st H.S. £150.
Sheffield Royal Hospital.—Ophth. H.S. Also Anæsthetist. £120 and £80 respectively.
Sheffield Royal Infirmary.—Asst. Aural and Ophth. H.S. and H.S. and Second Asst. Cas. O. Each at rate of £80.
Shrewsbury, Royal Salop Infirmary.—Cas. O. and Res. Anæsthetist. At rate of £160.
Stafford, Staffordshire General Infirmary.—H.S. £200.
Stockport Infirmary.—H.P. £175.
Swansea General and Eye Hospital.—H.P. £150.
Swansea, Swansea and Merthyr Tydfil Joint Mental Hospital.—Med. Supt. £800.
Taunton and Somerset Hospital.—Sen. H.S. At rate of £150.
Walsall General Hospital.—H.S. At rate of £150.
Weir Hospital, Grove-road, Balham, S.W.—Res. M.O. £150.
West London Hospital, Hammersmith-road, W.—Res. Asst. S. £200. Also H.P., H.S.'s, and Res. Anæsthetist. All at rate of £100.
Weston-super-Mare General Hospital.—Res. H.S. £150.
Winchester, Royal Hampshire County Hospital.—H.S. At rate of £100.
York, City Mental Hospital, Fulford.—Asst. M.O. £500.

Births, Marriages, and Deaths

BIRTHS.

FISHER.—On August 12th, at Welbeck-street, W., to Barbara, wife of Surgeon-Lieutenant H. H. Fisher, Royal Navy—a daughter.

GONIN.—On August 12th, at 53, Kew Bridge-road, Brentford, the wife of Dr. M. W. Gonin, of a daughter.

MARRIAGES.

BUCHANAN—ROBERTSON.—On August 15th, at St. Giles' Cathedral, Edinburgh, John Cecil Rankin Buchanan, M.D., M.R.C.P.E., East African Medical Service, to Eileen Isobel, elder daughter of Mr. and Mrs. J. D. Robertson, Redhurst, Ravelston Dykes, Edinburgh.

LORRAINE—HANSFORD.—On August 8th, at Great Bursted Parish Church, Essex, Norman S. R. Lorraine, M.D., Ch.B., D.P.H., F.R.S.E., to Joan, only daughter of Mr. and Mrs. E. C. Hansford, of Overstrand, Westcliff-on-Sea.

DEATHS.

DIXON.—On Sunday, August 16th, suddenly, at Whittlesford Cambridge, Prof. W. E. Dixon, M.A., M.D., F.R.S., LL.D., F.R.C.P.

FLACK.—On August 16th, Martin Flack, Group Captain, R.A.F. Medical Service.

JOHNSON.—On August 15th, Rance D. Johnson, M.R.C.S., L.R.C.P., of Heath Hurst-road, Hampstead, suddenly.

LACY.—On August 15th, at Wonston Cottage, Sutton Scotney, Hants, Charles Sethward de Lacy Lacy, M.A., M.B., in his 85th year.

N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages and Deaths.

NOTES, COMMENTS, AND ABSTRACTS

HOSPITALS AND MOTORING ACCIDENTS.

RECOVERY OF EXPENSES OF TREATMENT.

A MEMORANDUM[1] has been prepared by a barrister for the Central Bureau of Hospital Information explaining in non-technical language the effect of Section 36 (2) of the Road Traffic Act, 1930, in so far as it gives hospitals a claim on insurance companies for the expenses of treatment in hospital of the victims of motor accidents. The memorandum also deals with certain difficulties that have arisen in practice under the Act, and contains the answers to a number of inquiries that have been received.

It must be realised that Section 36 (2) of the Road Traffic Act is intentionally restricted in its effect, and that, large as is the number of victims of motor accidents who are treated in hospitals every year, insurance companies will only have to make payments to hospitals in respect of such treatment in a very small proportion of cases. This proportion is, in fact, estimated to be about 10 per cent.

HOSPITALS ENTITLED TO PAYMENT.

Payment for the expenses of treatment of victims of motor accidents can be claimed by any hospital that is "an institution which provides medical or surgical treatment for in-patients." The question has been raised whether this definition includes a hospital where it is usual only to provide nursing and accommodation, leaving the patients to arrange with their own doctors to attend them. The answer is that, so long as there is any medical staff who would ordinarily deal with emergency cases, the hospital comes within the definition. In the rare case where there is no such staff, the answer will depend on the judicial interpretation of the words "medical or surgical treatment"; in the meantime it is recommended that claims should be put in against insurance companies in all such cases.

CONDITIONS UNDER WHICH CLAIM CAN BE MADE.

The right of a hospital to obtain a payment from an insurance company for the expenses of treatment of the victim of a motor accident depends on the following conditions being fulfilled:—

(*a*) The victim must, as a general rule, be either (i) a person who was not in the car that was responsible for the accident, or (ii) a person who was a paying passenger in the car.

(*b*) The insurance company must have made a payment to the victim or his dependents in respect of his personal injury or death.

(*c*) The victim must have received treatment in the hospital as an in-patient.

(*d*) The hospital must not have made any charge in respect of the victim's treatment.

Even when all these conditions *are* fulfilled, the hospital will not *necessarily* be entitled to a payment from an insurance company. These conditions are merely the sine qua non for any such payment.

VICITMS IN RESPECT OF WHOM CLAIM CAN BE MADE.

It is only in respect of a very restricted class of victims that the expenses of treatment can be recovered by hospitals from insurance companies. The owner or driver of the car responsible for the accident will never come within the class. The car owner's friends or employees, being driven in the car, will come within this class when the policy has been specially so drawn as to include them, but not otherwise. If it is possible for the hospital secretary to see the policy issued by the insurance company in any particular case, he will be able to satisfy himself as to the exact classes of persons covered by the policy.

"A person not in the car that was responsible for the accident" of course includes any person in any *other* car that is involved in the same accident, as well as a pedestrian, a rider, &c. The question of responsibility for an accident is often most difficult to decide, especially when more than one car is involved in it, but fortunately, hospitals need not concern themselves with this point, as it will be decided for them. As, however, it will always be difficult for a hospital to know whether it has a good claim for the expenses of treatment of each individual patient, it is recommended that notice should be given to insurance companies concerned of every victim of a motor accident that is admitted to the hospital.

CASES IN WHICH HOSPITALS HAVE NO CLAIM.

In the following cases the victim will receive no payment from an insurance company, and consequently the hospital will not be entitled to any payment from the insurance company for the expenses of the victim's treatment.

(*a*) If the driver or owner of the car responsible for the accident is himself killed, or dies before his victim obtains a judgment for damages against him, or before his claim is otherwise settled.

(*b*) If the victim of the accident dies before his claim is decided, and he leaves no dependents who are entitled to claim damages for his death.

(*c*) When the accident has been caused by an uninsured person in a stolen car, or in a car he is not authorised to use.

(*d*) When the accident has been caused by a car owned by a local authority or a police authority, by an invalid carriage—i.e., specially designed for use by a person suffering from a physical disability—or by a tram car or trolley vehicle the use of which is authorised by special Act of Parliament or Order

PAYMENT BY INSURANCE COMPANY TO VICTIM OR DEPENDENTS.

Is it necessary to prove the negligence of the car driver before the hospital can recover a payment from the insurance company? The answer is that *the hospital* is not concerned with any question of negligence, but must merely ascertain whether any payment to the victim or his dependents has in fact been made by the insurance company under its policy; if such a payment has been made, then the insurance company must also make a payment to the hospital in respect of that victim's treatment. In point of fact the insurance company will only make this payment to the victim either (i) if it is agreed, or has been decided in court, that the car owner or driver insured by this company was responsible for the accident, or (ii) if responsibility is not proved or admitted, but the insurance company, in order to avoid legal proceedings, makes a payment to the victim by way of a compromise of his claim—called a "compassionate grant."

As the recovery by hospitals of expenses of treatment from insurance companies depends on the victim having actually received some payment in respect of his injury, it may possibly be worth the while of hospitals to consider whether they could obtain the honorary services of a local solicitor to look into the claims of victims (being patients in hospital) who either neglect, or have not the means, to take the necessary steps for pressing their claims themselves.

EXPENSES OF TREATMENT THAT CAN BE RECOVERED.

The claim of a hospital against an insurance company is limited to £25 for each patient. Subject to this, the hospital can claim, at the rate of the average cost of an occupied bed per day, for the number of days that the victim has been treated as an in-patient. The average cost of an occupied bed per day is arrived at by taking the hospital's

[1] Copies on application to the Director of the Bureau, 12, Grosvenor Crescent, London, S.W. 1. 3*d*. post free.

total annual expenditure on the maintenance of the hospital and its staff, and on the maintenance and treatment of patients, and dividing this figure by the total of daily counts of in-patients.

Claims for payment can only be made in respect of in-patient treatment. No extra payment can be claimed for an operation or for any special form of treatment. There appears to be an idea that the Act would allow a hospital to pay its medical staff special fees for attending motor-accident patients, and to increase the costs to be recovered from an insurance company by the amount of such fees. It is quite clear that the Act does not allow of this being done. The costs per day to be recovered for a motor-accident patient must be the same as the average cost for *all* the patients in the hospital.

CHARGES MADE BY A HOSPITAL FOR TREATMENT.

The condition that "the hospital must not have made any charge in respect of the victim's treatment" gives rise to several difficulties, and raises the question as to what payments hospitals can receive from victims without losing their claim against insurance companies.

(*a*) *Voluntary payments by victim.*—It seems clear that a purely voluntary payment by a victim to the hospital cannot legally prejudice the hospital's claim for a subsequent payment from the insurance company. Whether it is quite fair to insurance companies that hospitals should receive both payments is another matter, and this question might well be considered when some general agreement between hospitals and insurance companies is being negotiated.

(*b*) *Invitation to pay part of cost of treatment.*—In hospitals where the usual practice is to invite patients to make such payment as they are able towards the expenses of their treatment, the question has been raised whether the acceptance of such a payment from the victim of a motor accident could be considered as "making a charge." Although the matter is not free from doubt, it is thought that such a payment may safely be treated as a voluntary payment; but care should be taken that the payment is expressly made as a "donation."

(*c*) *Practical difficulty of deciding whether to make a charge or not.*—When it is the practice in a hospital to make a definite charge to a patient of what it is thought he can pay, it is now difficult to decide how to proceed in motor accident cases.

The hospital is in a dilemma; if it makes any charge to the victim while he is in hospital, it loses its claim for a payment by the insurance company—and this payment *might* be considerably more than the victim could pay; but if it makes no charge for treatment while the victim is in hospital, and if, after the victim has left, it turns out that the hospital has no claim against the insurance company, then it will probably be too late to recover anything from the victim.

There appears to be no way out of this difficulty at present, and it can only be solved by some general agreement with insurance companies. One suggestion, which appears to be an equitable solution, is that the hospital should obtain what payment it can from the victim while he is in hospital, and deduct the amount of this payment from its claim against the insurance company. Hospitals might perhaps endeavour to come to an agreement with insurance companies on these lines in individual cases, pending the negotiation of a general agreement.

(*d*) *Payments to hospitals under certain contributory schemes.*—If the victim of an accident is treated in hospital, and the hospital claims a payment for such treatment out of the funds of a contributory scheme to which the victim subscribes, the question has been raised whether this claim must be considered as a "charge for treatment," and so preclude the hospital from obtaining a further payment from the insurance company. This again appears to be a subject on which some general agreement with insurance companies should be reached.

METHODS OF EVASION.

Some insurance companies are attempting to evade part of their liability under the Road Traffic Act. In some cases, an insurance company, before settling the victim's claim, has tried to bargain with a hospital as to its claim for expenses of treatment, and to persuade the hospital to accept less than the full amount to which it is entitled. Insurance companies have no right to do this, as the method of calculation of what they must pay to hospitals is clearly stated in the Act. In other cases, an insurance company has agreed to pay a definite sum to the victim under the policy, and has subsequently deducted from this sum the amount of the payment made by the insurance company to the hospital. Strictly speaking, this does not concern hospitals. The matter must be settled between the insurance companies and the victims. In other cases the insurance company announces to the hospital that it is proposing to make an ex gratia grant to the victim, but will only make the grant if the hospital undertakes not to demand any payment from the insurance company for the expenses of the victim's treatment. Such a threat by the insurance company is most improper.

The practical difficulty in all these cases is that if hospitals stand on their legal rights, it will probably result in insurance companies being less generous in the settlement of the claims of victims; and if this becomes apparent, it will obviously have the effect of raising a prejudice against hospitals, as it will be said that the hospitals are receiving payments which ought to have been made to the injured. Probably the only satisfactory method of settling these questions will be by a general agreement with insurance companies.

PROCEDURE IN MAKING CLAIMS.

Whenever the victim of a motor accident is brought to a hospital, information should at once be obtained from the police as to the registration numbers of all cars involved in the accident, the names of their owners, and the insurance companies by whom they are insured against third-party risks. A notice should then be sent to the insurance company—or to each insurance company, if more than one car is involved. As soon as the victim leaves hospital, or, if he has to stay in hospital for a long time, as soon as the expenses of his treatment (calculated at the average daily cost of an occupied bed in that hospital) amount to £25, a formal claim for the expenses of treatment should be sent to the insurance company. The memorandum suggests the phrasing of these notices of claim.

ALCOHOLIC CRAVING.

IN the course of a paper on the Alcohol Habit, read to the Paddington Medical Society on August 11th, Dr. ALEX. BALDIE discussed the nature of the craving for alcohol, which was bound up with that of the craving for all drugs of a habit-forming tendency. Its causes might, he thought, be fairly if briefly summarised as follows:—

ITS CAUSES.

(1) The chemical influence exerted by the drug on metabolism—an influence for good or ill which is liable to become more specific and indispensable as tolerance is established.

(2) The conscious contrast between the cerebral relaxation induced by alcohol and the emotional release consequent on such relaxation, and the mental lassitude experienced when the effect of the drug has passed.

(3) Automatism. Repetitive acts, whether whistling, humming a tune, scratching the head, biting the nails, interminably lighting and puffing a cigarette, or calling for drinks, all equally act as diversions to a mind preoccupied. An act repeated becomes a habit, the habit a vice, and the vice a destiny.

(4) Misinterpretation of natural thirst and fatigue.

To describe the nature of the habit in other words he quoted Trotter ("Instincts of the Herd in Peace and War"): "Almost universally regarded as either, on the one hand, a sin or vice, or, on the other hand, as a disease, there can be little doubt that in fact (the alcohol habit) is essentially a response to a psychological necessity. In the tragic conflict between what he has been taught to desire and what he is allowed to get man has found in alcohol a sinister but effective peacemaker . . . . a way out of the prison house of reality."

This escape from reality—that is, from emotional and intellectual tension—or relaxation, is an easy process normally for a good many people. We all know the fortunate ones who are able at will to relax either into slumber, as for 40 winks in an armchair, or into gaiety and irresponsibility after strenuous mental work. This process, easy and habitual to a favoured few, is impossible without the aid of narcotics to the vast majority of mankind. Hence the desire for such aid, and—too often—for its immoderate use. Hence also the misunderstandings and the differences of opinion as to the necessity for its use, and the irritation engendered by propaganda and legislation directed towards its restriction.

FATIGUE A CONTRIBUTORY FACTOR.

Alcoholics tire easily, as do other debilitated persons. Periodic rest at short intervals tends to prevent the sense of fatigue which is a contributory element in the craving to which they are subject. Even in normal civilised persons the sleep function is sufficiently abused. A continuous period of 16 hours of wakeful activity in the perpendicular attitude, followed by habitual slumber for eight hours in the horizontal, does not appear physiologically the most desirable habit. It has no parallel amongst the mammals at any rate. The tired nervous system of the chronic alcoholic responds quickly to a recuperative régime—such as, for workers, a short diversion and physical relaxation every two or three hours. This need not be for more than about ten minutes. It should be accompanied by the regular drinking of a variable quantity of liquid. Also complete relaxation for about an hour, after the mid-day meal, should be encouraged. It is unfortunate that the conditions of modern life, which provide abundant toilet facilities in public places, and opportunities everywhere for exercise, recreation, and refreshment, should provide none for rest; so that an individual miles away from his home, as many are compelled by reason of their occupation to be, cannot obtain rest and privacy, short of the somewhat expensive and eccentric process of taking a room in a hotel for an hour. Lucky are those exceptional persons who are able, at will, to steal 40 winks in a crowded tube train or on a hard seat in Trafalgar-square. The facilities afforded in this respect until recently by the silent film have been considerably curtailed by the advent of the "talkies."

ITS REDUCTION.

No treatment of chronic alcoholism can be effective which does not include a deliberate attempt to strengthen the will of the patient to discard the drug and to understand and undermine the potency of the craving which is his besetting sin, vice, and disease. This is the part of the treatment which is difficult and complex since it must be approached on special psychotherapeutic lines; but here, as in other directions, the family doctor, by virtue of his intimate knowledge of him, may be able to guide and control his patient's diseased tendency. The craving may not necessarily be simple in its origin—few cravings are—but an understanding of possible causes may suggest a simple and practical method of treatment.

(1) The craving may be due in part to the established and specialised influence on metabolism which we know alcohol possesses. To this extent its replacement by other forms of fuel, such as sugar, may be of use.

(2) The unrelieved overactivity of the higher mental faculties, from the strain of which alcohol has provided an habitual escape, must be avoided and another escape discovered. In other words, outlets for the expression of repressed emotion should be cultivated, in the form of recreations, interests, and pursuits most adapted to the physical and psychological personality with which we have to deal.

(3) The alternations of high and low blood pressure induced by the alcoholic transformation should be borne in mind. Generally depression and despair go with a low blood pressure; energetic and high-spirited optimism with a high. It is in its effects in this direction that exercise and occupation, not fatiguing and preferably of some useful, interesting, and remunerative kind, is indicated.

(4) The not unimportant influence of the repetitive and automatic nature of the drinking ritual requires attention. The mental preliminaries to "another little drink" may be active against the conscious will and knowledge of the patient, who must be alert to understand his unconscious tendencies and to break the repetitive chain. "Substitution" habits may be objectionable, but may help.

(5) Misinterpretation of simple thirst will be avoided if the patient assuages his natural thirst periodically with small doses of aqua pura. Misinterpretation of fatigue will be prevented by periodic rest.

ITS PREVENTION.

The majority of civilised mankind, said Dr. Baldie, uses alcohol moderately and with benefit. But the considerable minority who through idiosyncrasy, foolishness, or excess is unable to consume it safely or beneficially requires something more than exhortations and threats if it is to be influenced. Probably there is scope for yet another "temperance" society, which shall be free from all ulterior motive, and which will help ignorant persons to understand the pros and cons of alcoholic indulgence, and to know how, when, and where to stop. The following suggestions for the rational indulgence in alcoholic liquors might be of help to such a society:—

(1) Drinks should be avoided invariably between meals.
(2) They should not as a rule be used to assuage excessive thirst.
(3) They should be avoided generally during mental or physical exertion.
(4) They should be avoided absolutely under all conditions requiring the exercise of skill, judgment, and technical or mechanical efficiency.
(5) All non-teetotallers should imbibe by the clock small quantities of fresh water, since some part of the alcoholic craving is unconsciously due to natural thirst, and natural thirst is not to be assuaged by anything but pure water.
(6) The wheat should never be mixed with the grape, the use of spirits should be restricted to meal times, of beer to that of a beverage under normal mental and physical conditions, and wines to social occasions when it is desired to relax the psycho-motor inhibitions.
(7) Solitary indulgence on social and psychological grounds should be discouraged as tending to cause secret drinking and tippling.
(8) As corollary to (7) all bona fide social reformers should encourage the revival and extension of facilities for legitimate indulgence in restaurants, theatres, and other such places, devoted to the alimentary, social, and recreational requirements of the public.

Present Incidence of Alcoholism.

As a matter of historical fact it would appear that alcohol reaches its greatest popularity and use in times of stability and prosperity. Clinical experiences suggests (1) that the general incidence of alcoholism is slightly less than formerly, though not to the extent represented by some propagandists; (2) that the habitual use of alcohol is diminishing to a greater extent; and (3) that the social offence of drunkenness—i.e., inebriation of a disorderly or incapable type—has declined considerably.

FACTORS OF DIMINUTION.

The factors of diminished habitual alcoholism may be tabulated as follows:—

(1) Lessened popularity. Owing to increased mental tension, a relatively short period of exhilaration

or relaxation is produced by alcohol in the present as compared with the Victorian non-addicted population. The tendency in the case of the present generation is to experience more intensely the irritability, discomfort, and confusion of the second stage of inebriation; there is on this account, and to this extent, less habitual drinking. On the other hand, in times of stress and change, mental conflict becomes more intense and universal. This and other factors have widened the range of susceptibility to alcohol, and its use has remained almost as general, though it is relatively less habitual than formerly.

(2) The heavy and prohibitive taxation of alcoholic beverages and other restrictions.

(3) The shorter hours of work and the higher standard of living enjoyed now by that section of the public from which the statistics of old-time police-court drunkenness were drawn.

(4) Increased facilities for young persons for amusements, games, recreation, and adventure—the last being a very important item.

(5) A loosening of conventional restraint between the sexes, and the outlet provided by the freer intercourse thus possible.

(6) The extension of education, and of knowledge concerning the disadvantages of alcoholic excess.

FACTORS OF PERSISTENCE.

The factors which combine to cause the persistence of habitual alcoholism of a morbid degree are:—

(1) Group pessimism and restlessness, due to insecurity and conflict caused by (*a*) economic difficulties—e.g., unemployment, high taxation, &c.—(*b*) the disturbance of social and moral outlook, particularly in sexual and religious spheres.

(2) The encouragement of habits of secrecy and excess through indulgence according to opportunity rather than to necessity, caused by the restriction of hours and places in which alcohol may be openly sold and consumed.

(3) The relatively large increase in drinking amongst women, consequent upon the greater independence and freedom exercised by the sex.

(4) The absence, in Great Britain and Ireland, of amenities such as music and dancing, and of facilities for the use of alcohol socially, in public and with meals, such as abound elsewhere.

(5) The group urge towards individual discipline, sacrifice, and sustained and urgent effort.

(6) The increasing organisation of local transport and movement in the body social and economic, without adequate provision for physiological rest and privacy for the individual.

(7) The inability of most persons to relax at will.

(8) The biological necessity or craving for play and other escapes from reality, and the inadequacy of modern life to provide such escapes.

(9) The social, moral, and political antagonisms which are produced by all biological deprivations compulsorily applied, reasonably or unreasonably, by the group to its members.

THE WATER OF SWIMMING BATHS.

THE outdoor bather in Switzerland is fortunate in having the choice of abundant clean water in many natural lakes. Where he is thrown back on the swimming pool he is faced with the dangers that are now pretty well recognised in this country. A recent writer in the *Gazette de Lausanne* remarks that the tanks of popular swimming baths contain "a veritable purée of bacteria," the numbers of which may reach 40,000 colon bacilli and 130,000 other germs per cubic centimetre. He insists on the special danger of such water to the conjunctiva of the diver and of the exponent of the crawl and other modern strokes involving repeated immersion of the head, as evidenced by the acute follicular conjunctivitis which is peculiar to frequenters of swimming baths, and which appears after six to ten days incubation. The pathogenic agent of this disease is a filter-passing virus, and the clinical and histological aspect of the malady resembles that of trachoma. Although it may last for two to three months the affection however is not serious. The nasopharyngeal mucosa is also often affected by the polluted water of swimming baths. One observer has published statistics of 90 cases of otitis, tonsillitis, rhinitis, laryngitis, pseudo-membranous anginas, and furunculosis of the external meatus, all observed 24–36 hours after swimming in a bath. Another has recorded, in three years, a dozen cases of otitis in "crawlers."

The causes of these dangers are not far to seek, but the writers of this article and of another in the *Feuilles d'Hygiène et de Médecine*, which appears to have inspired it, do not seem to be aware of the success which has been obtained by filtration, followed by chlorination. It is very properly pointed out that filtration is insufficient, and that the sediment consisting largely of desquamated epidermis containing billions of bacteria per cubic millimetre requires a strong dose of chlorine to sterilise it. The plan of removing easily filtrable and coagulable matters and then chlorinating is not discussed. Until a preliminary cleansing with soap and brush can be insisted upon in public as it is in some private baths, the author advises swimmers not to dive or practise strokes *tête dans l'eau*. Finally, the sound advice is given not to confound cleanliness and sport, to wash in washing baths, and when clean to swim in swimming baths. One might have hoped such advice would be redundant, but even in England one has seen rather dirty boys in swimming baths. In the United States of America catchy slogans have been used with some effect.

THE PURPOSE OF AN EXPERIMENT.

A MEDICAL man, who is a director of the Scottish Society for Prevention of Cruelty to Animals, has called our attention to an advertisement which has appeared several times lately in the *Scotsman*, sent by the hon. secretary of an anti-vivisection society, and quoting from THE LANCET of May 24th, 1930 (p. 1120), the following experiment performed by Dr. G. W. Theobald in the course of work on the nature of eclampsia.

"Bitch, near term; 45 c.cm. of a 4 per cent. solution of potassium oxalate were injected intravenously in three doses of 10, 15, and 20 c.cm. between Jan. 7th and 10th, 1929. The anus was closed on the 9th and the bitch was put on a meat diet. On Jan. 11th the animal refused food; on the 12th it reeled around as if drunk, and on the 13th it went into convulsions. The back was extended, the eyes remained open, and there was a marked tremor of the limbs. The bitch died after the convulsions had lasted one hour and before the pups were born."

The advertisement invites readers to help to stop cruel experiments by subscribing to the funds of the Society. No direct comment is made on the particular experiment, but a reader who saw the advertisement wrote to the *Scotsman* to say: "I regard anti-vivisection as a wrong fad which stands in the way of scientific progress . . . but I cannot see the lesson to be gained from the class of experiment" quoted from THE LANCET. We have asked Dr. Theobald to set out in a few sentences what the experiment was devised to prove, and he replies: "True eclampsia has never been known to occur in a dog. The object of the experiment was to cause intestinal stasis by mechanical means and to lower the blood calcium by the best known non-operative method—i.e., by the injection of potassium or sodium oxalate. The clinical symptoms produced were indistinguishable from eclampsia, and could be relieved by the intravenous injection of calcium chloride." Dr. Theobald adds that the spectacle of a women suffering from eclampsia and the suffering she endures are appalling.

Corrigendum.—The analysis of the death-rate from diabetes mellitus in the United States, annotated in our issue of August 15th (p. 364), appeared in the Statistical Bulletin of the Metropolitan Life Insurance Co. of New York.